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THE NEUROLOGIC ASPECTS OF NASAL SINUS INFECTIONS

HEADACHES AND SYSTEMIC DISTURBANCES OF NASAL GANGLION ORIGIN *

SIMON L. RUSKIN, M.D.

NEW YORK

In considering the neurologic aspects of nasal sinus infections, one is confronted by clinical pictures remarkable in both their variety and their extent. Many of the manifestations which have been attributed to the absorption of toxic products from the infected sinuses are not necessarily such and can be definitely traced to direct nerve irritation from the inflamed sinus membrane. One gets not only symptomatology from the direct influence of the diseased area on the nerve filaments supplying it, but remote effects through pathways that at first glance appear impossible but which in careful analysis show a fairly direct route from the diseased mucous membrane. It is the purpose of this paper to direct attention to this avenue of disturbance which is equally deserving of consideration with that of the influence produced on the system by the absorption of toxic products.

Physicians have all been more or less accustomed to thinking of the evil effects produced in the stomach by the swallowing of nasal secretions from an acute or chronic sinusitis, or of the damaging effects of chronic nasal discharges in the respiratory tract, which induce pharyngitis, laryngitis, bronchitis and even bronchiectasis; however, the influence on these same organs through chronic nerve irritation induced by the inflamed sinus mucosa on the vegetative nervous system has hardly received the attention it merits.

The neurogenic disturbances arising from nasal sinus infections can be grouped as: (1) acute pains in sinus inflammation, (2) chronic neuralgias, (3) functional vasomotor and secretory states and (4) systemic effects induced chiefly through the vegetative nervous system.

The nerve tracts through which these influences are effected are the trigeminal system, the facial nerve system and the vegetative nerve system. The influence induced through the trigeminal system has received

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* Read before the Philadelphia College of Surgeons, March 12, 1928.

* The anatomic studies presented in this series are intended to demonstrate, in a practical way, the great amount of mucous membrane covering the area of the nasal sinuses. One sees at a glance the many possibilities for the retention of irritating secretions, as well as the manifold sources of irritation of the nerve.

wide consideration, especially by Sluder, whose work is as classic as it is beautiful. I have tried to emphasize especially the symptomatology arising from the facial nerve and vegetative systems.

When one considers that developmentally the nose in the lower animals is probably the greatest factor in the preservation of the species with a wide variety of physiologic functions, ranging from the regulation of temperature to the recognition of the presence of its enemies, one is not surprised to find that nature has massed its most important nerve tracts there superficially, and established important connections. The success of its adaptation is dependent on the reflex as well as on the direct effects of nasal stimuli. In the nasal mechanism the olfactory nerve probably does not play a more important rôle than the stimulation of the trigeminal, facial and vegetative systems. The animal scenting a change in environment reacts reflexly with its eyes and ears and its sympathetic and endocrine systems. The ammoniac odor of the urine of a dangerous enemy calls into instant play not only the epinephrine mechanism of the sympathetic system, but also the facial expressions used by some animals to strike fear into their enemies, as well as an increase in acuteness of hearing and sight and the sensitiveness of the face, thus bringing to play all three nerve tracts.

In man, this same mechanism, utilizing these developmental tracts, can arise not necessarily from the stimulation of the nose by vaporized substances, but by disease processes stimulating the same fundamental tracts. I have also seen cases in which certain odors will induce, in sensitive persons, attacks closely simulating the entire picture. The analogy that I have drawn may at first sight seem far fetched, but careful clinical observation will tend to bear this out more and more. Olfactory stimulation is probably coincident with stimulation of the other nasal nerves. A noxious odor is not only recognized as such through the olfactory fibers, but is reflexly reacted to by the nasal ganglion and ethmoidal branches of the trigeminal nerves. So one sees nasal engorgement and rhinorrhea following the inhalation of chlorine. Mild odors, of course, do not produce such marked symptoms, but there is stimulation in proportion to the dilution of the vaporized substances. Salivation, so intimately associated with smell, is also related to the nasal ganglion through the nervus intermedius of Wrisberg.

The reflexes of the nose have long had a reputation for mysterious peculiarity and inexplicability. Their pathways, however, are the three systems which I have mentioned.

THE TRIGEMINAL SYSTEM

The trigeminal nerve has its great ganglion, the gasserian, within the cranial cavity. Leaving it are the three great trunks—the ophthalmic to the eye and nose; the maxillary to the nose, cheek and mouth, and the

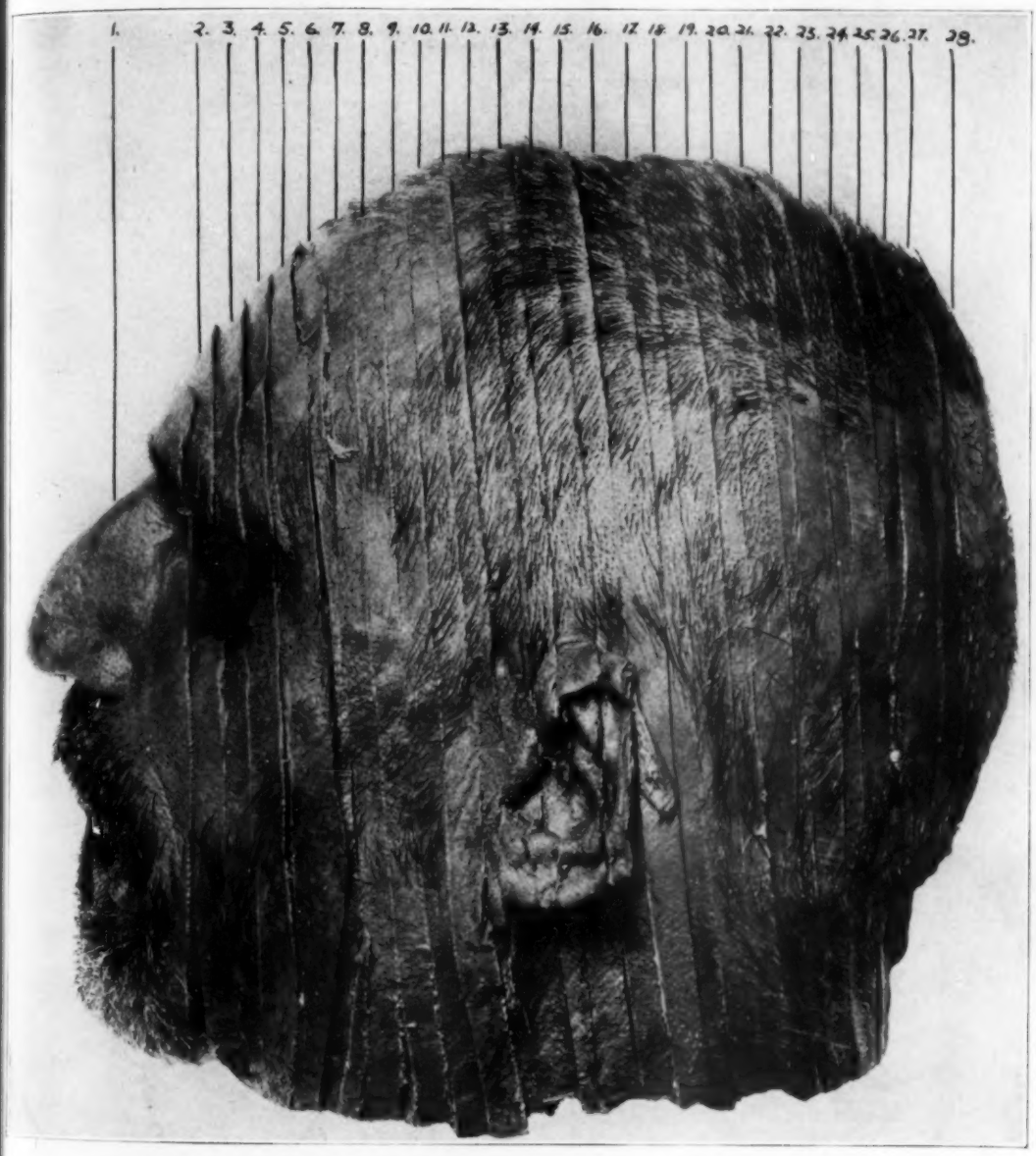

Fig. 1.—Serial sections of the head (Ruskin Anatomical Museum).

mandibular to the mouth and lower jaw. Each of these large trunks has slung on it a small ganglion possessing sensory, motor and sympathetic fibers. The ophthalmic trunk has the ciliary, the maxillary, the sphenopalatine, the mandibular and the otic. Developmentally, the trigeminal

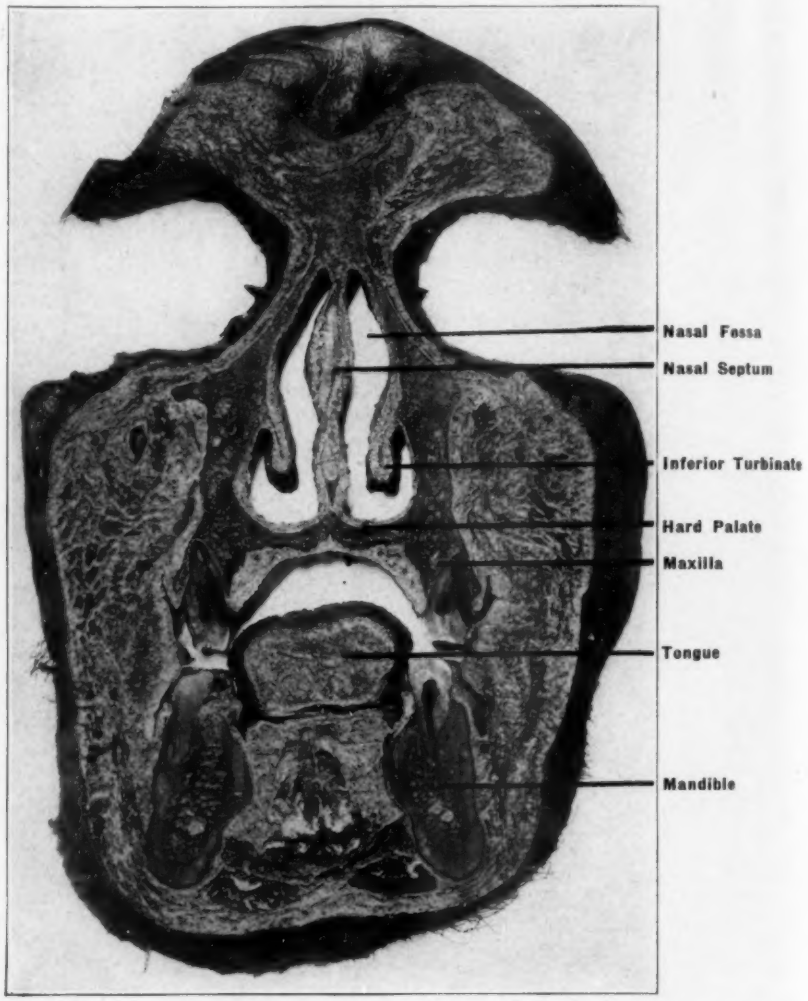

Fig. 2.—Note the thickening of the septum area formerly cauterized for dysmenorrhea by Fleiss.

system has probably had the chief function of adjusting the animal to its environment, with the three ganglions acting as reflex centers for rapid visceral reactions. All three ganglions are connected with each other and have direct connection with the vegetative nervous system. Though these ganglions one gets the sympathetic manifestation of fright,

and I have frequently been able to induce it by irritation of the sphenopalatine ganglion. In the routine nasal treatment one occasionally elicits this syndrome, and the patients will inform one that they feel unduly frightened. If I may digress, the frequency of a history of

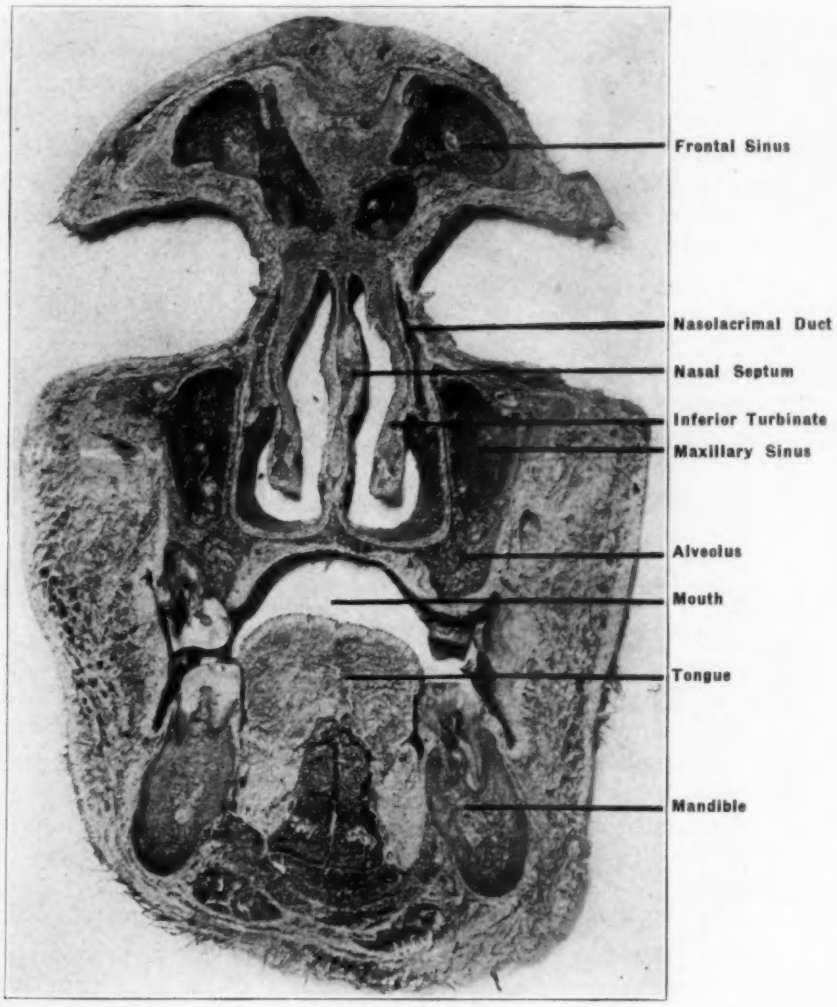

Fig. 3.—Note the irregularity of the mucous membrane lining; small cysts.

so-called "nervous breakdown," meaning usually a state of continuous fright, is strikingly frequent in cases of nasal sinus infections. These three ganglions were probably the first line of defense, and reflexly served more quickly than cortical action. The sphenopalatine is the largest and most influential of the three ganglions. It is significant that

the sympathetic filaments from the cavernous plexus run to the ciliary ganglion, those from the internal carotid and superior cervical sympathetic to the nasal ganglion and those from the middle meningeal to the otic ganglion. It is therefore not surprising that one can profoundly affect the sympathetic system through the nasal ganglion. Here in the nose is the only sympathetic nerve center that is placed superficially, exposed to disease processes and within easy access for treatment and experimentation. This is the only place where the sympathetic system can be reached without operative intervention, and should yield rich results in the research of the vegetative nervous system.

The branches to the nose from the trigeminal system are the two or three sphenopalatine nerves running from the maxillary division to the sphenopalatine ganglion. From here the branches are distributed in four groups: a descending group contributing the palatine nerves to the roof of the mouth, palate and upper two thirds of the tonsils, a lateral nasal group to the lateral wall of the nose, throat and sinuses and some branches extending into the orbit, a medial group to the septum and a posterior group to the sphenoid. All these areas are a possible source of irritation to the trigeminal nerve. In addition, there are the anterior ethmoidal and posterior ethmoidal—both branches from the ophthalmic division of the trigeminal nerve—supplying the sinus and septum areas in addition to the external nose.

I shall now consider the circumstances under which irritation to the nerves will arise. Strangely enough, a suppurating sinus, after the acute stage has subsided, will apparently produce but little disturbance to the nerves. It is chiefly after a lapse of time, generally years after the acute sinusitis, that the customary neuralgic syndromes are found. It is in the type of case in which the roentgen report reads, "Slight thickening of the lining membrane, no evidence of fluid," that one finds the neuralgic syndromes. Some rhinologists classify these cases as "hyperplastic ethmoiditis, sphenoiditis, maxillary sinusitis," viewing them as distinct entities. In opening these sinuses on anatomic material, I have observed that gross changes in the mucosa are frequently associated with viscid secretions or multiple small cysts, and I have gained the impression that these hyperplastic states have been induced by the presence of secretion lying in the sinus for a long period until absorbed by the mucous membrane, and in the same process, inducing a chronic thickening of the membrane, as well as a low-grade neuritis of the nerve endings in the mucosa.

The clinical pictures of involvement of the trigeminal system can be grouped practically under the three main branches. The involvement can be direct as in the ophthalmic migraine secondary to irritation of the ethmoidal nerves which are direct branches of the ophthalmic division,

or indirect from the nasal ganglion, traveling by way of the maxillary nerve to the gasserian, and there involving filaments of the ophthalmic division. In this manner, one has to deal with ophthalmic neuralgia, passing usually under the term migraine, or ophthalmic tic, maxillary

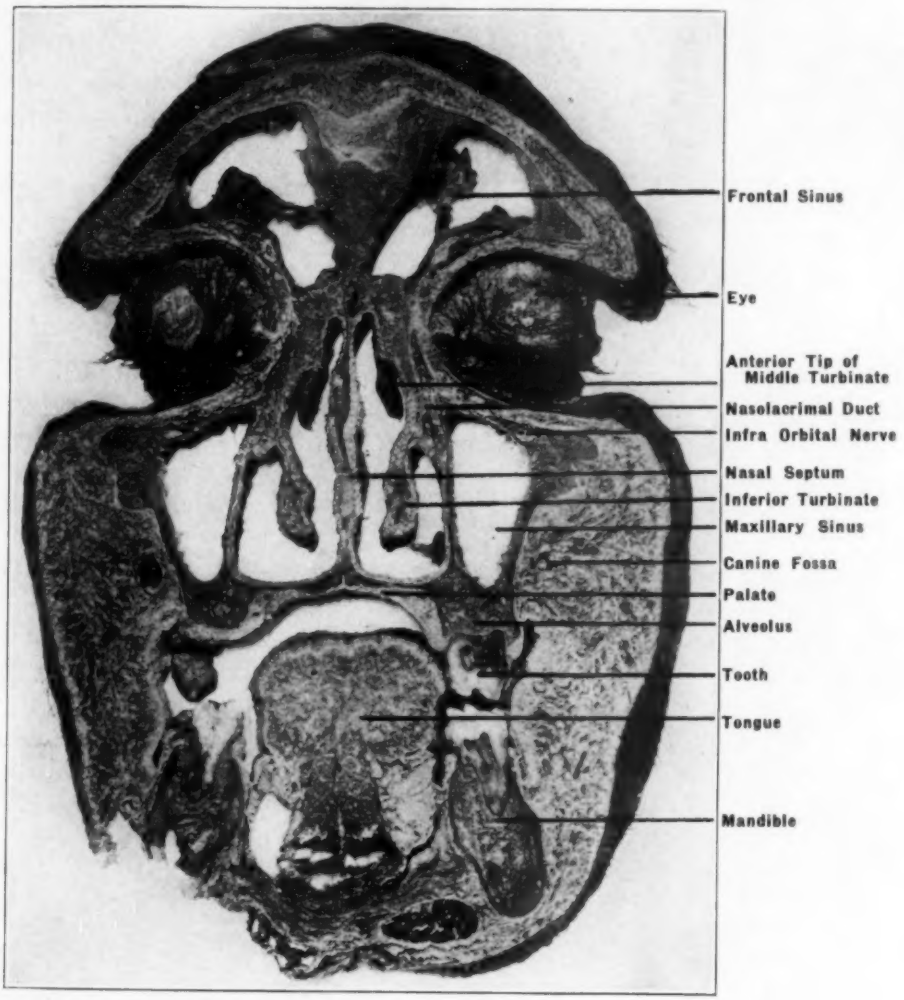

Fig. 4.—Note how the eye is practically surrounded by the nasal sinuses.

neuralgia, mandibular neuralgia or combined trigeminal neuralgia with or without tic douloureux.

To illustrate briefly I shall describe a case of each.

True Ophthalmic Neuralgia.—This condition frequently goes under the common name of migraine. Dr. B., aged 41, came to me in February, 1926, with the diag-

nosis of migraine. He weighed 200 pounds (90.7 Kg.), was 6 feet in height, was robust, and had complained for the last four years of pain beginning in the left eyeball, sometimes supra-orbital or infra-orbital, and radiating to the vertex and, occasionally, to the mandible. He had no radiation to the back of the head. The

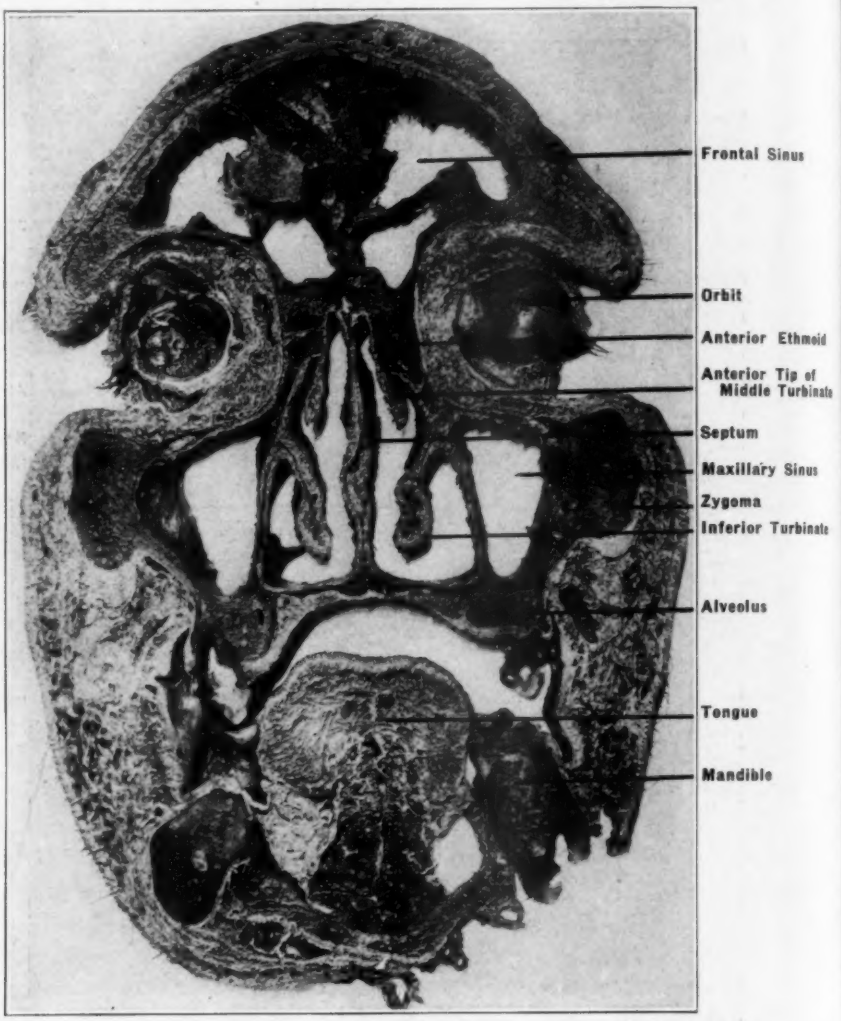

Fig. 5.—Note the great extent of the frontal sinus, and the large amount of mucous membrane exposed in the sinuses in this section.

attacks were sharp and associated with nausea. They were induced by sneezing, coughing or laughing. He had some remissions in warm weather. In his history it is significant that he had influenza ten years ago. The roentgenograms of the sinus were negative except for slight thickening of the membrane. Nasal observations were practically negative. Antrum washing had been performed previously with negative return.

Local treatment of the nasal ganglion gave immediate relief. This was the first real relief he had experienced in the four years of his complaint. After a series of eight local treatments, he had been comfortable until the time of writing.

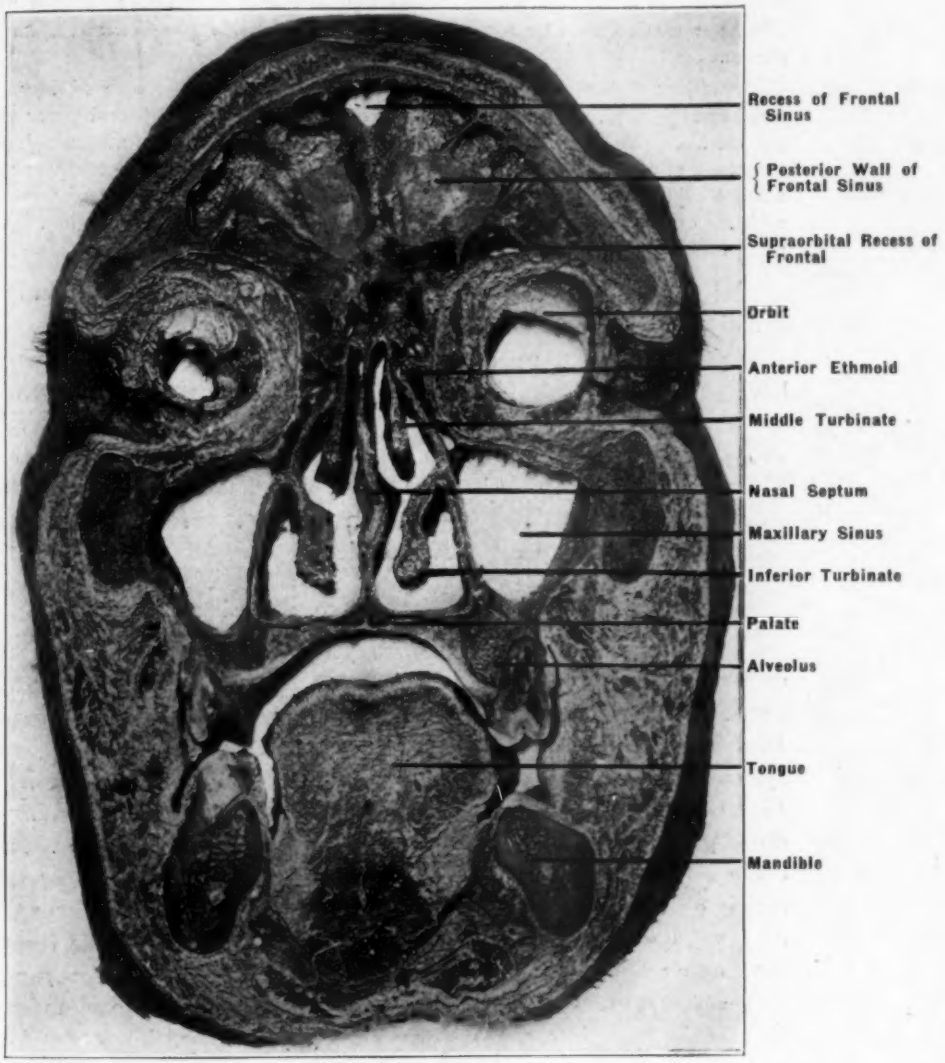

Fig. 6.—Note the many recesses of the frontal sinus, and the small ethmoidal cells.

Ophthalmic Tic.—Equally interesting is the case of Mr. S., who had suffered from ophthalmic tic; that is, whenever he touched the tip of his nose a sharp stabbing pain would shoot into his eye and dart into his temple and then to the vertex of his head. The condition in this case responded to local applications made to the anterior and posterior ethmoidal nerves. In another patient with ophthalmic

migraine, Madame D., the condition responded to local applications to both the nasal and the ethmoidal nerves. She had suffered for the last forty years from recurrent attacks of frontal pain of such severity as to cause her to strike her head against the wall during attacks. The injection of alcohol into the supra-orbital nerve, as well as the intra-orbital injection into the ethmoidal nerve, was without avail. The pain was relieved by local applications to the nasal ganglion and ethmoidal nerves.

Maxillary Neuralgia.—This condition is illustrated by the case of Mrs. W., a woman, aged 65, who had suffered from pain in both antrums and upper gums for the last twelve years. Nasal examination showed a moderate atrophic rhinitis. Roentgen examination revealed slight thickening of both antral membranes; there was no evidence of pus. She had undergone the most varied forms of treatment with no effect. Injections into both maxillary nerves and nasal ganglion by the palatomaxillary canal route gave a splendid result.

I shall omit mandibular neuralgia since it is most frequently of dental origin, and is relieved by blockage of the mandibular nerve.

Entire trigeminal neuralgia and its relationship to tic douloureux, I have described in a previous paper. I wish merely to mention that tic douloureux seems to be the simultaneous involvement of the trigeminal and facial nerves, and I have shown that the nasal ganglion receiving fibers from both systems is what I may call the trigger for the attack. The cases I have previously reported indicate the excellent results obtainable by injections into the nasal ganglion.

THE FACIAL SYSTEM AND THE NOSE

I shall now leave the trigeminal system and turn to a consideration of the facial nerve system and the nose. Developmentally, the facial nerve serves a much greater rôle in the lower animals than it does in man. In fishes it takes over, to some extent, the function of the trigeminal system. Here it plays prominently a sensory function. In the higher forms it is an integral part of the protective mechanism, innervating the muscles of the face in a manner to drive terror into its opponent. Especially significant is the action of the muscles surrounding the eyes, the action of the orbicularis oculi in narrowing the palpebral fissure, the corrugator supercili in bringing the eyebrows together and the frontalis in bringing the eyebrows down in a menacing manner. The same muscles also constitute what are called the accessory muscles of accommodation; they are brought into play whenever the eyes are used intently and produce the expression of peering. The same mechanism works in accommodation. The interesting thing is that, in chronic irritation of the nasal mucosa with irritation of the facial nerve, this same mechanism is called into action as in the instance of stimulation of the nose in the lower animals in the search for food or detection of their enemies. As a result of the unnecessary overaction of these accessory muscles of accommodation, a distinct headache characterized frequently as "ocular

headache" develops in these patients. Whereas the stimulation of the facial nerve mechanism in the lower animals by food or enemies is only temporary, that induced by disease processes in the nose is continuous,

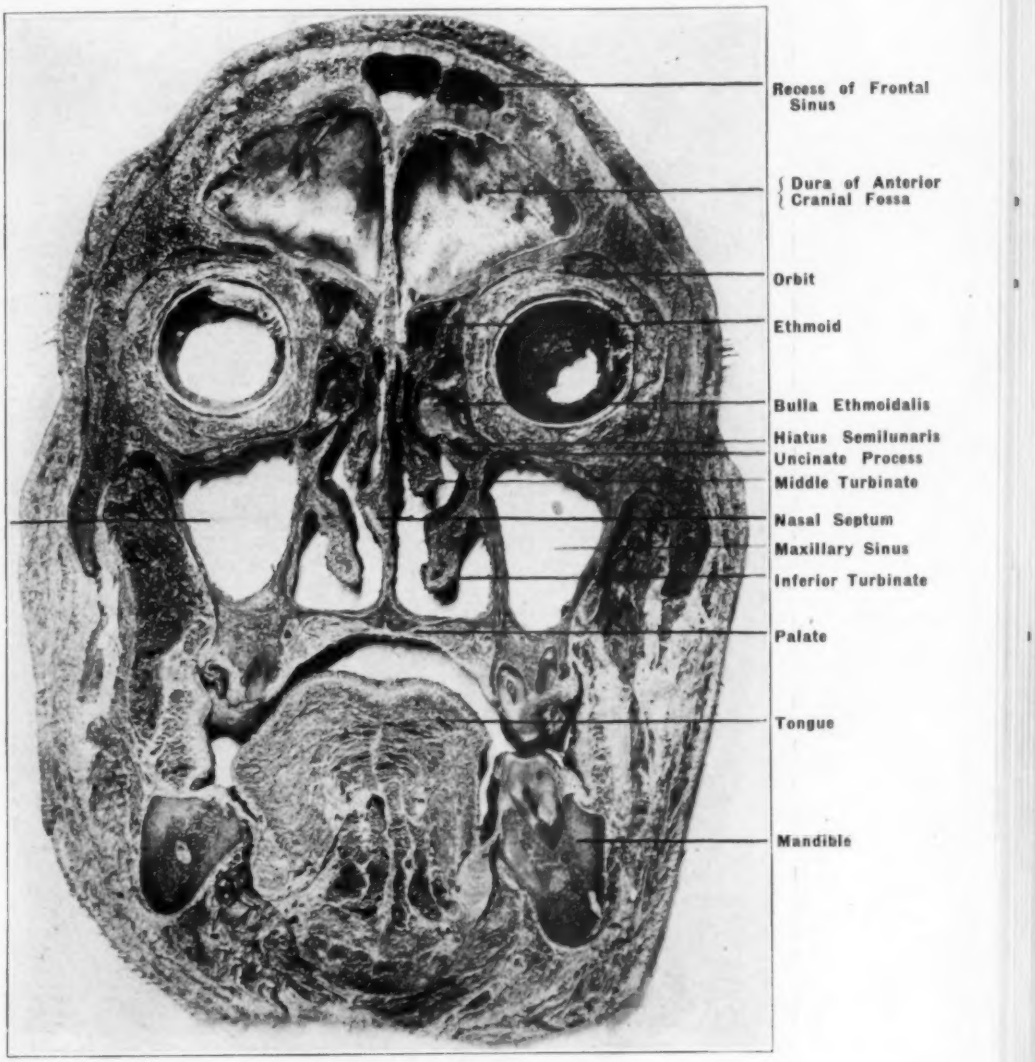

Fig. 7.—Note the relatively large size of the maxillary sinus.

and exhausts the patient physically and mentally. These patients have a frown characterized by a small vertical wrinkle at the head of each eyebrow. Observations on the eye are consistently negative and glasses produce practically no improvement. The eyes feel heavy, tired and uncomfortable. The pain across the back of the head, which the patients

complain of, is caused by the pulling forward of the scalp by the frontalis muscle. These conditions frequently are encountered under the diagnosis of "asthenopia." In these cases, blockage of the nasal ganglion

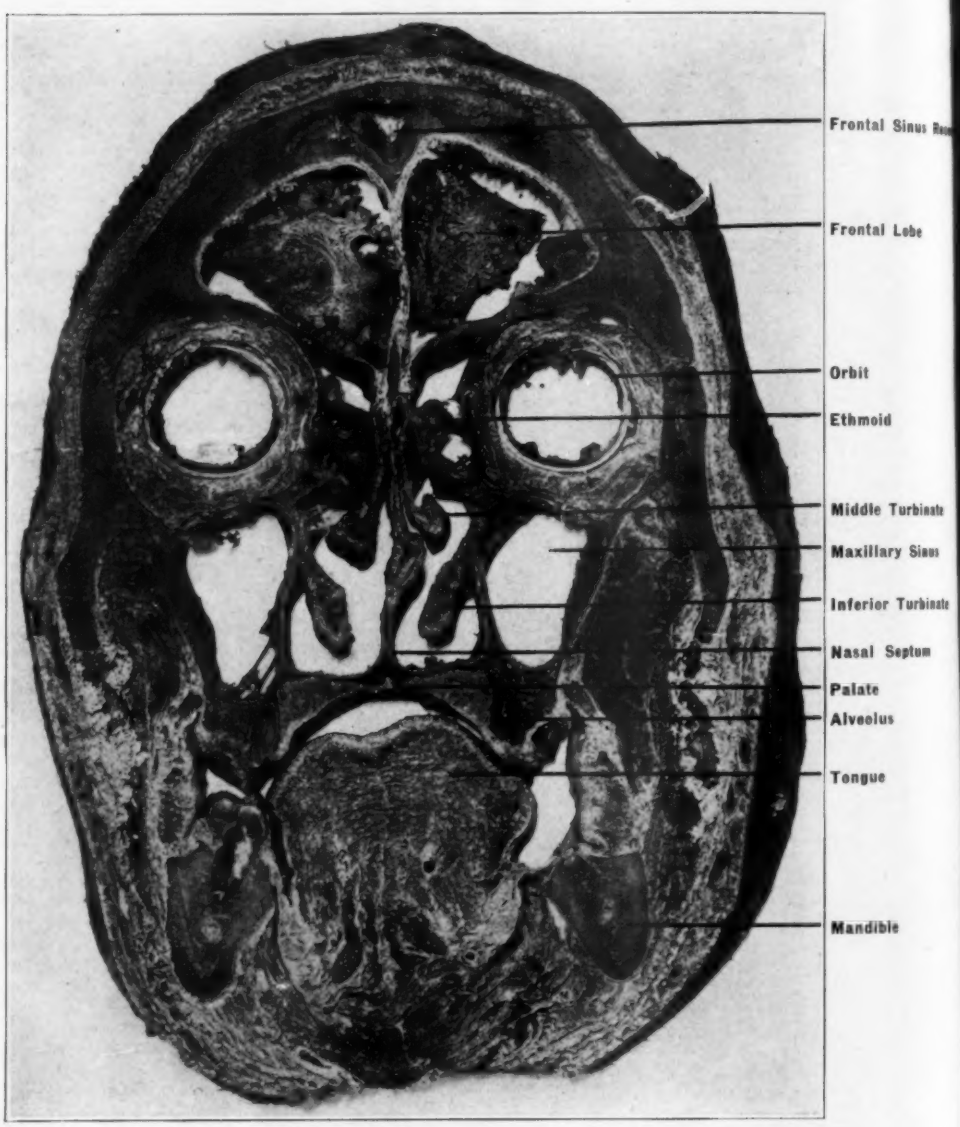

Fig. 8.—Note the increasing size of the ethmoid sinus.

produces considerable relief. The overaction of the accessory muscles of accommodation relaxes, the eye is rested and the patient has a feeling which is described "as a feeling as though the eyes were washed."

I have given this description as a new explanation for so-called ocular headache of nasal origin, and to indicate the relationship of the facial nerve.

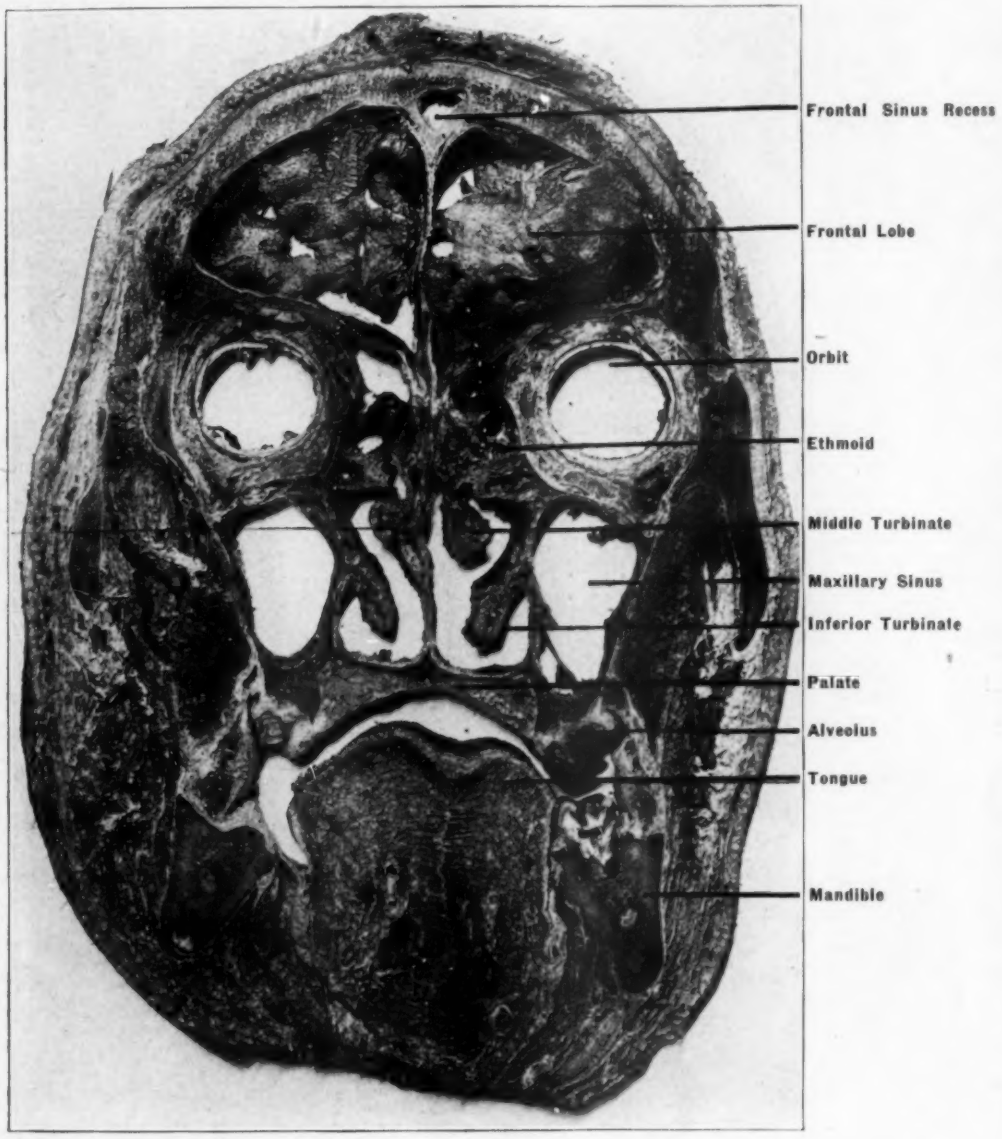

Fig. 9.—Note the many ethmoidal recesses as well as the frontal recess.

I shall now go back to a careful consideration of the facial nerve. One usually thinks of the facial nerve as a motor system. The motor facial, however, travels with and is intimately associated with the

nervus intermedius of Wrisberg, a sensory nerve that, according to Hunt, is the sensory portion of the facial nerve. The nervus intermedius, which I shall refer to as a sensory facial, has the geniculate

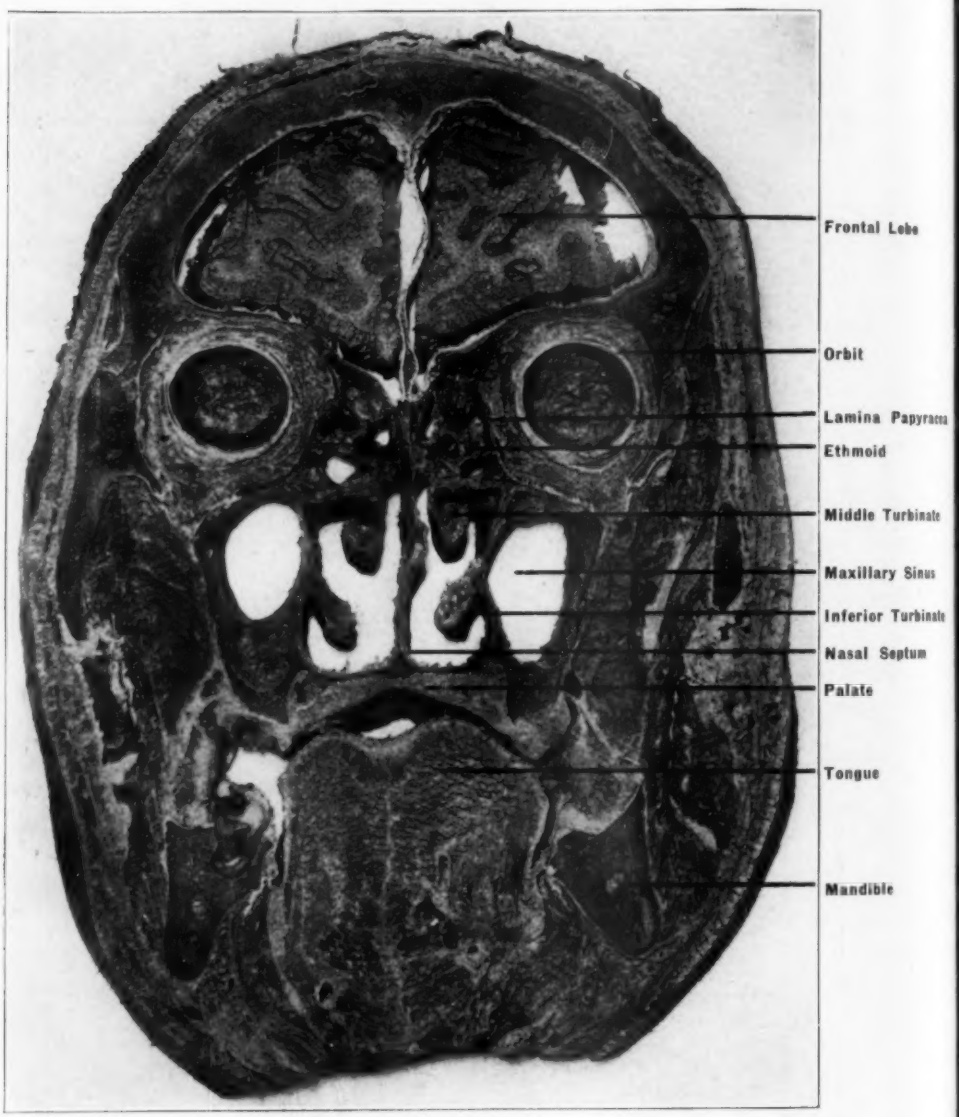

Fig. 10.—Note the many cells of the ethmoid and the area of mucous membrane exposed.

ganglion which is situated on the motor facial nerve, where the motor facial makes its bend at the medial tympanic wall to run posteriorly toward the antrum of the mastoid, carrying with it branches from the

sensory facial, which later leaves it as the chorda tympani. From the geniculate ganglion itself to the nasal (sphenopalatine) ganglion runs a fairly large nerve called the greater superficial petrosal. It is this latter

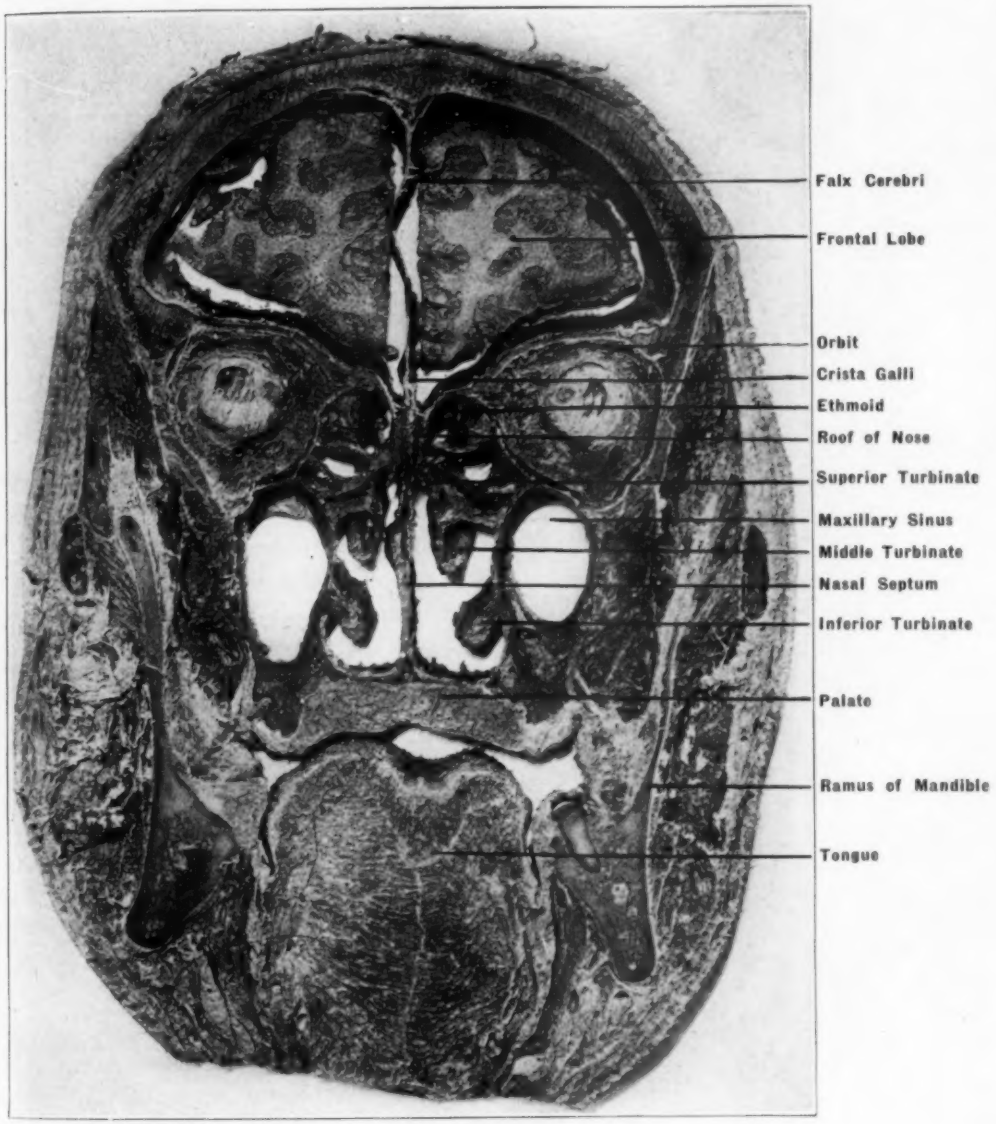

Fig. 11.—Note the relationship of the retro-orbital structures.

connection that is of such great importance to physicians. The character of the fibers of the greater superficial petrosal is a matter of considerable interest. It has been generally conceded that taste fibers pass by way

of this nerve to the nasal ganglion, from there to be distributed to the roof of the mouth, side of the mouth and tongue. I have proved this clinically in my earlier publications, wherein I recited the case of Mr. K.,

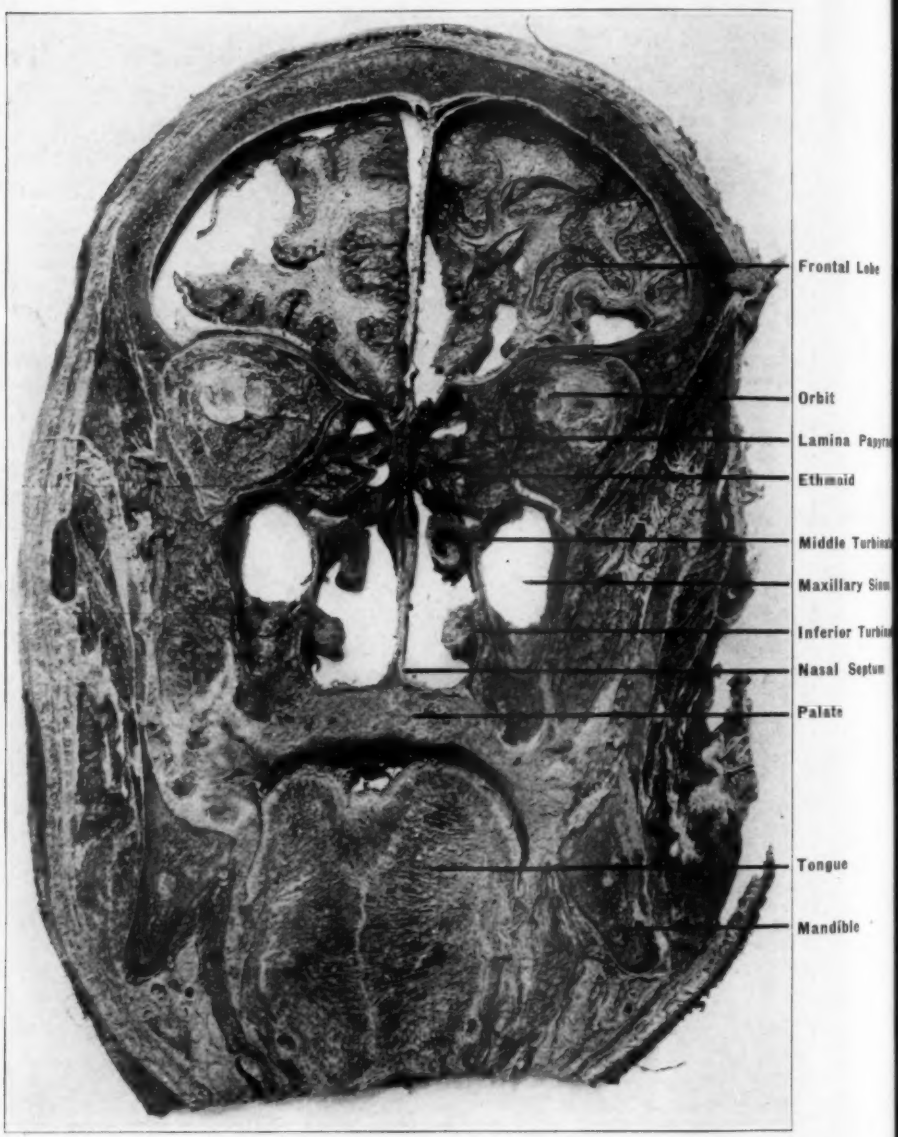

Fig. 12.—Note the plastic exudate in the maxillary sinus.

who suffered from disturbances of taste and a burning sensation along the side of the mouth and the tip of the tongue, which were relieved by blockage of the nasal ganglion with alcohol. Tear fibers of cranial

autonomic origin are claimed to occur in the greater superficial petrosal and to go on to the lacrimal gland by way of the nasal ganglion. I have also proved this academic claim by first observing a partial drying of the tear secretion as a coincident factor in some of the cases in which I had made injections into the nasal ganglion for neuralgic purposes and then by injection into the nasal ganglion in a case of severe epiphora, which resulted from stenosis of the lacrimal canal, following an old unsuccessful dacryocystectomy. In the latter case, there was a distinct diminution of the tear secretion. In my article on "the sensory field of the facial nerve" I suggested injection into the nasal ganglion as a new method for the control of excessive tearing. Since, however, the lacrimal gland has a double innervation, the tear secretion cannot be completely affected in this manner. I have also pointed out the common observation of diminished secretion of tears on the side affected with Bell's palsy. Motor fibers to the levator palati have been described in the greater superficial petrosal. Hunt described herpes of the roof of the mouth, faucial pillars and uvula traceable to fibers from the greater superficial petrosal, hence implying the presence of sensory fibers. In my paper, "Contributions to the Study of the Sphenopalatine Ganglion," I pointed out that those pains usually described as part of the "Sluder Syndrome" of nasal (sphenopalatine) ganglion neuralgia referable to the back of the head follow closely the distribution of the facial nerve rather than that of the trigeminal. If one glances at the distribution of the facial nerve according to Quain, it is found that communications are formed with the following:

1. At the internal acoustic meatus with the acoustic nerve by a twig which, according to Arnold, runs back from the geniculate ganglion. This could possibly explain the vertigo so frequently complained of in nasal ganglion neuralgia.
2. The sphenopalatine ganglion by way of the greater superficial petrosal.
3. The otic ganglion by a branch which joins the lesser superficial petrosal nerve.
4. The sympathetic fibers on the middle meningeal artery.
5. The auricular branch of the vagus.
6. The glossopharyngeal.
7. The vagus.
8. The great auricular.
9. The auriculotemporal.
10. The lesser occipital.
11. The cutaneous cervical.
12. The trigeminal.

The symptomatology arising from the nasal ganglion follows closely the distribution outlined for the back half of the head. This, in conjunction with the fact that there are cases in which there is either a

distinctly front half headache in nasal disease or a distinctly back half headache, has led me to suspect that I am dealing with two mechanisms: one a trigeminal, and the other a facial.

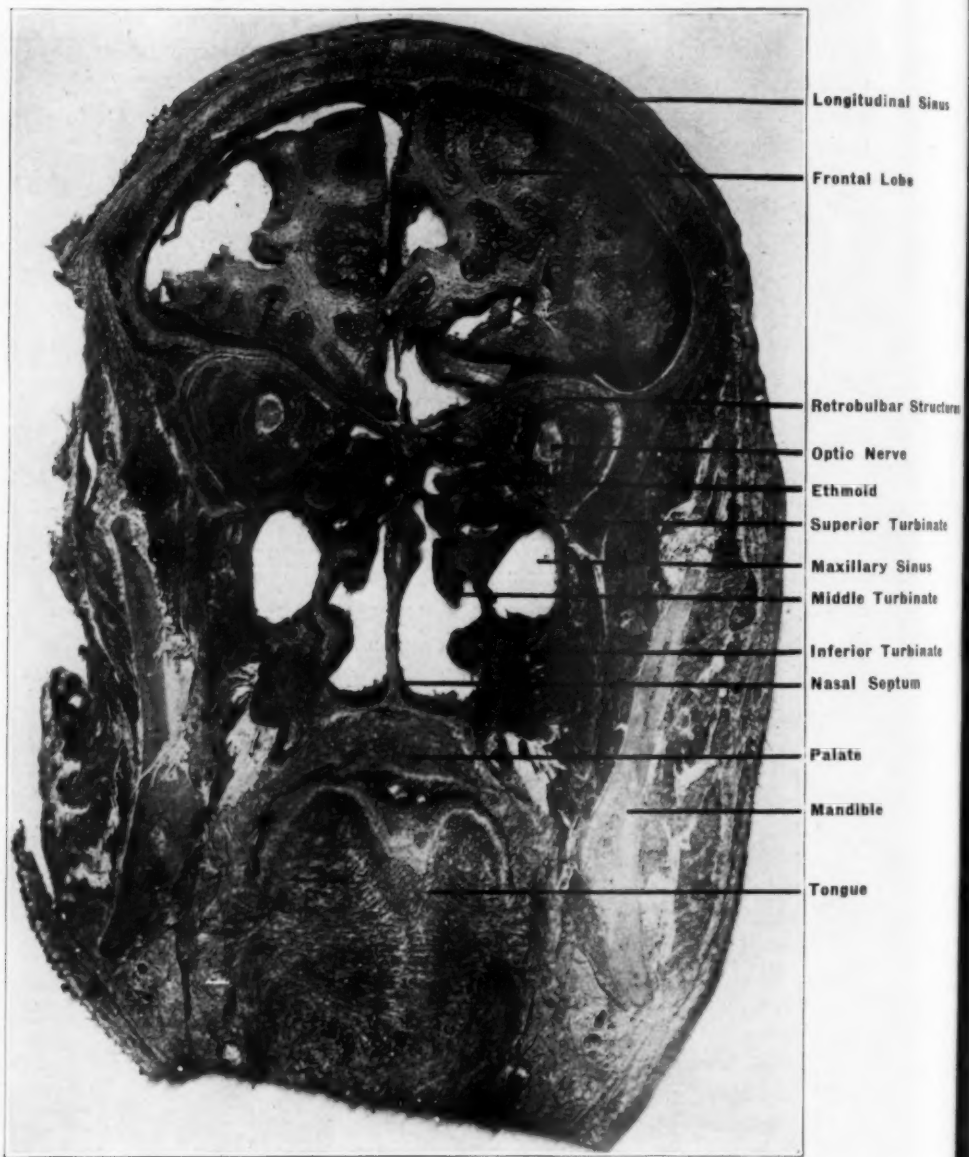

Fig. 13.—Note the ethmoidal cells in the same plane.

There is, however, a good deal more to this question of headache than merely a matter of distribution. There are two different forms of pain. The pains in the front half of the head are usually typical sensory

pains of trigeminal origin; those in the back half of the head are myalgic, muscle pains. The sensation is that of stiffness and soreness, comparable to rheumatic pains; frequently there are distinct myalgic nodes along the

Fig. 14.—Note the posterior wall of the maxillary sinus behind which lies the sphenomaxillary fossa.

origin or insertion of the cervical muscles frequently along the sterno-cleidomastoid and especially at the origin at the mastoid tip. This is a

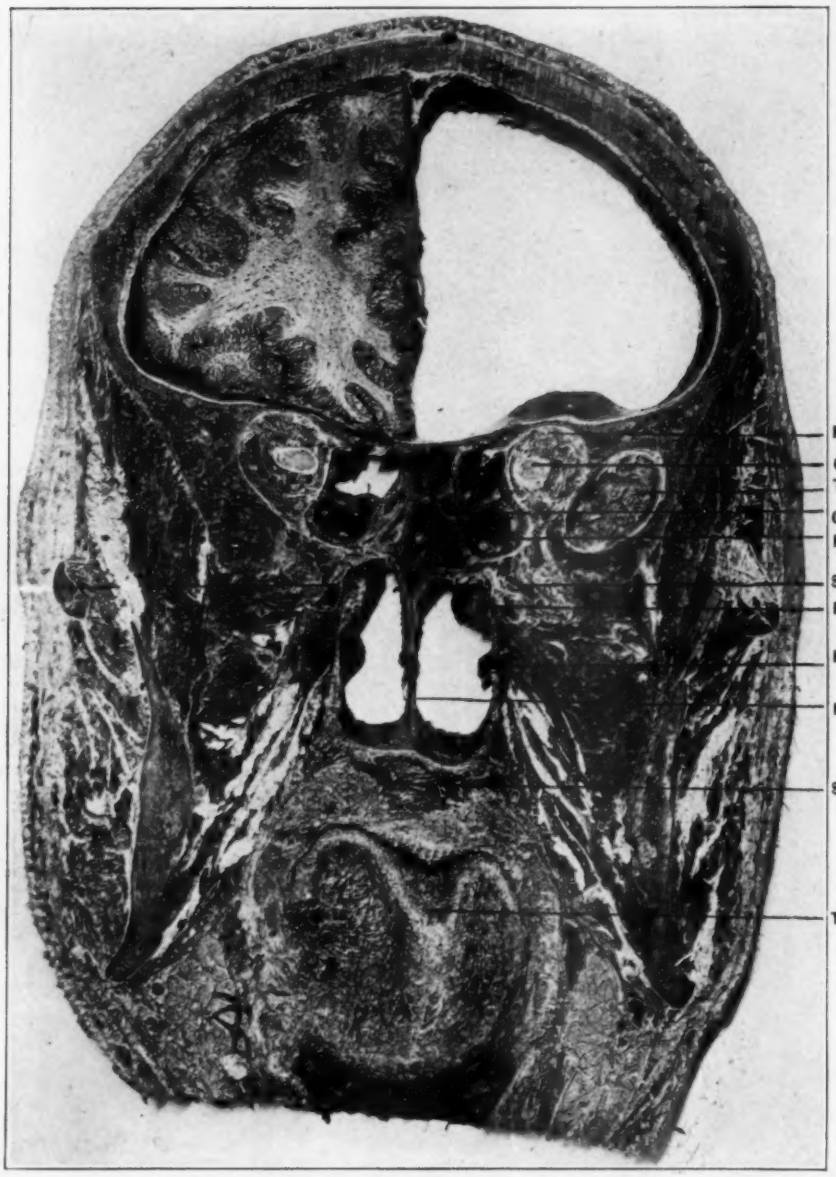

Fig. 15.—Observe the relationship of the sphenopalatine ganglion district to the sphenoid, ethmoid and antrum.

distinct condition, different from trigeminal pains. This is also the site of tenderness described as being behind the mastoid. These muscle attachments are tender to pressure. Halle considered this a distinct entity, but the interesting thing is that cocainization of the nasal ganglion quickly relieves this myalgic state and practically dissolves the nodes in a few minutes. I have a patient, Miss Bessie B., a slender girl, on whom these nodes stood out visibly on the trapezius and sternocleidomastoid muscles, appearing with her headache and disappearing with relief from the headache. Halle attempted the treatment of such myalgic nodes by severe and painful massage. This process is entirely unnecessary. What now becomes of the analysis of this back half headache? Merely this: Along the distribution of the facial nerve and its communication, one finds painful states, chiefly myalgic. These painful states are readily relieved by cocainization of the nasal ganglion, and were, therefore, classified by Sluder under the heading of the nasal ganglion neuralgia. Actually, one is influencing at the sphenopalatine ganglion the terminal fibers of the greater superficial petrosal nerve through which the facial nerve is reached. I also proved this by stopping the pain in the ear in herpes zoster oticus in two cases by cocainization of the nasal ganglion.

I shall now consider the question of these myalgic nodes and painful muscles of the back of the head. Here one is dealing probably with a mechanism that is neither sensory innervation nor motor innervation, but a vasomotor affair. Müller showed that the nervus intermedius carries cranial autonomic fibers, some of which pass to the nasal ganglion. I believe that cranial autonomic fibers also go with the branches of the facial fibers to the muscles, and that irritation of the cranial autonomics of the nose induces a vasomotor spasm along its distribution, similar to a pain along the course of a sensory nerve. This vasomotor spasm interferes with the circulation of the muscle and its metabolism, allowing an accumulation of lactic acid and throwing portions of the musculature into a low-grade rigor, at the same time creating a vicious cycle; the rigor itself there induces vasomotor disturbance. This would also explain the relief accorded by vigorous massage, diathermy and heat of various sorts. I shall also indicate later how easily the cranial autonomics are affected at the nasal ganglion, which explains the almost miraculous relief from these muscle pains. Not only can the autonomic distribution along the pathways of the facial nerve be affected, with the production of head pains, but a more diffuse effect on the involuntary nervous system can cause similar states in other muscles of the trunk and extremities. Sluder suggested that the pain in the neck, shoulder, arm and forearm is a manifestation of the involuntary nervous system, probably the sympathetic. This mechanism which I have described can be used to

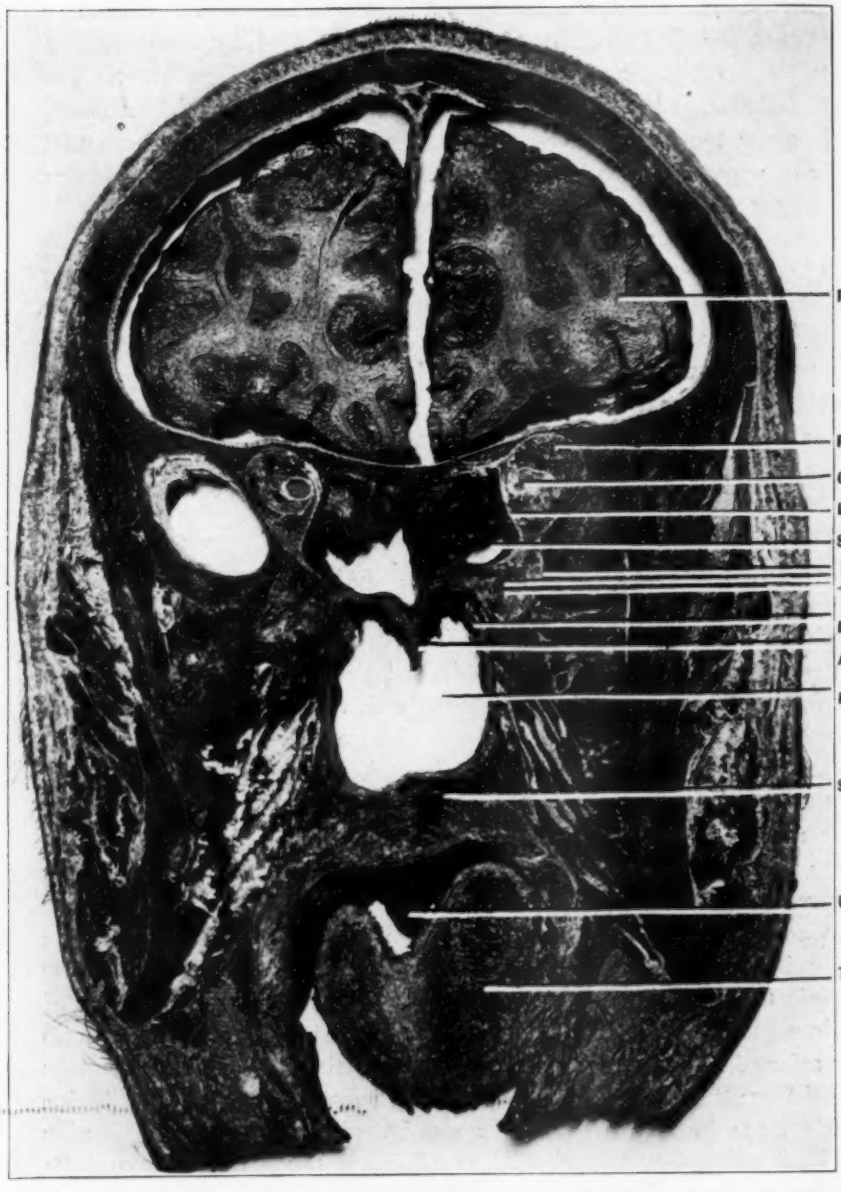

Fig. 16.—Note the fascia enveloping the retrobulbar structures and the sphenopalatine ganglion.

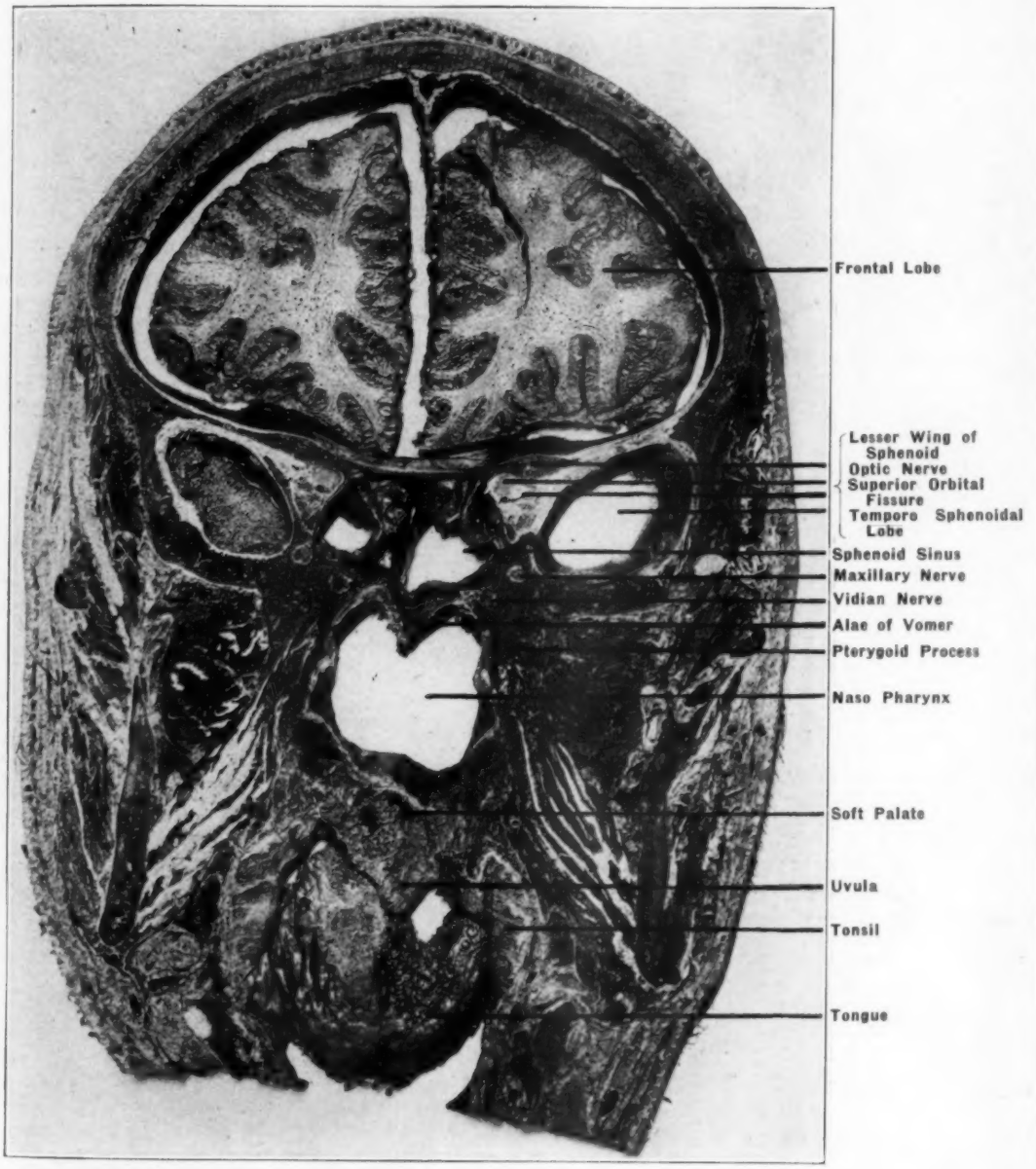

Fig. 17.—Observe the maxillary nerve and vidian nerves—both roots of the sphenopalatine ganglion.

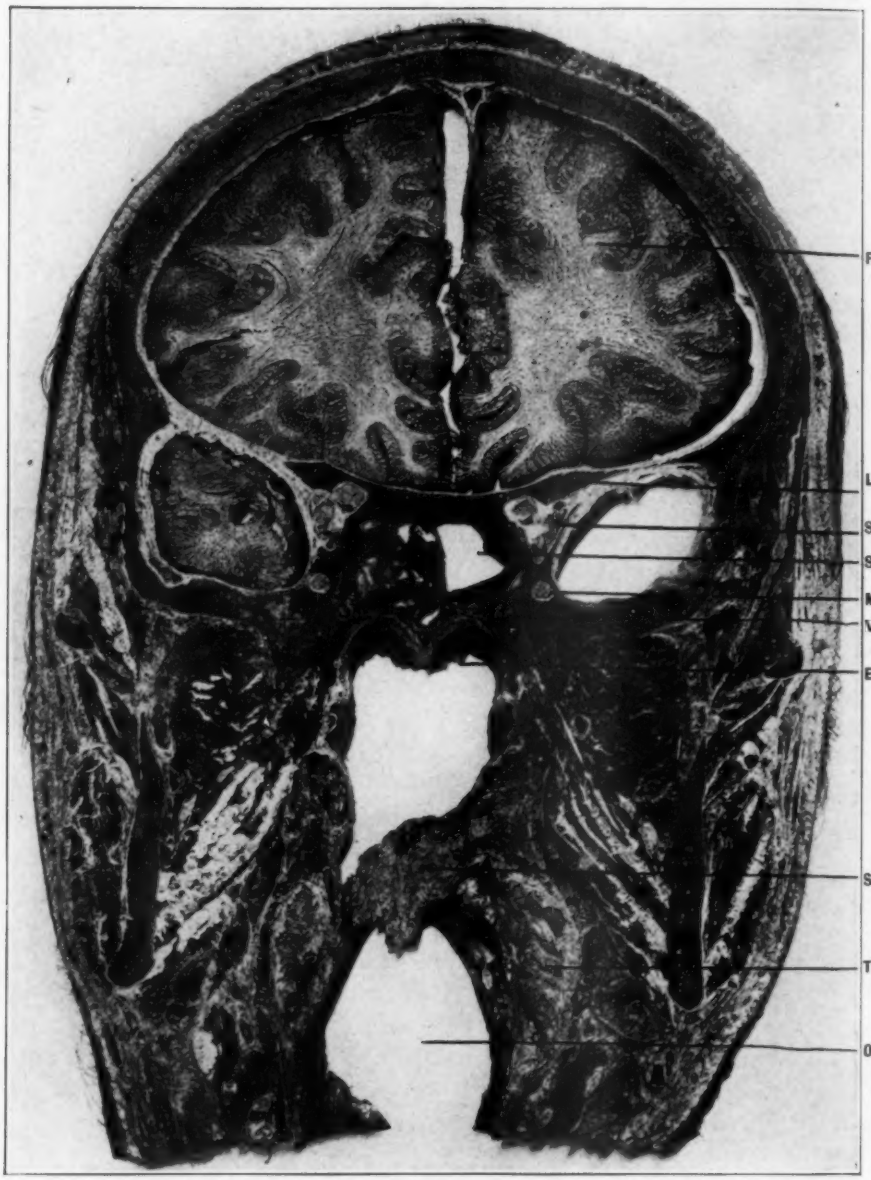

Fig. 18.—Note the intimate relationship between the nerve structures and the sphenoid sinus.

explain the almost uncanny results obtained in cases of severe pains of the shoulders and arms, lumbago and sciatica. When this vasomotor disturbance affects the diaphragm, persistent hiccup is produced. I have the case of Dr. L., who suffered for three days from severe persistent hiccup. I saw him on the third day, at which time he was receiving inhalation of carbon dioxide gas every two hours, with only temporary results. Following a single application to the nasal ganglion, he was completely relieved. For some time, for fear of it being branded psychologic and otherwise, I have hesitated to reveal the fact that I could, in a few minutes, relieve some patients from severe so-called sciatica which is really a vasomotor muscle disturbance. The patients stay free from such pains for a few years after three or four nasal treatments. Such patients seem to do better after local nasal application than after injection.

A similar muscle mechanism can also be induced in the front part of the head by way of the facial motor nerve fibers through continuous low-grade motor stimulation, as I have already indicated in regard to the muscles of the face, especially to the accessory muscles of accommodation with the resultant periorbital discomfort and headache. Frequently, in addition, the patient will complain of a peculiar stiffness of the cheeks or tightness across the bridge of the nose. The patient complains often of a pinching sensation at the root of the nose, or pains in the nasal bones. These pains are different from trigeminal neuralgic pains; they are low-grade discomforts, similar to lumbago pains. They are not connected with the trigeminal system but are located in the trigeminal area because the facial nerve supplies the same area. Cushing observed that in cases in which there has been a total resection of the gasserian ganglion, some patients still complain of low-grade discomfort in the face. I venture to say that the discomfort is due to the mechanism along the facial nerves, and that the trigeminal neuralgia is possibly of nasal origin and, perhaps, could have been relieved by injection of the nasal ganglion, with results similar to those I have attained in tic douloureux.

It is also interesting that patients approaching the menopause, at a time when their endocrine and vegetative nervous systems are undergoing transition periods, when there is a certain lability of the vasomotor apparatus, suffer from these same myalgic nodes and myalgic headaches, which are easily relieved by affecting the vasomotor apparatus through the nose. Another mechanism that is, apparently, of similar character is the choking sensation in the throat which I have found frequently associated with nasal ganglion neuralgias as well as with climacteric conditions. These are usually dispatched with the phrase "globus hystericus." This condition clears promptly with cocainization of the nasal

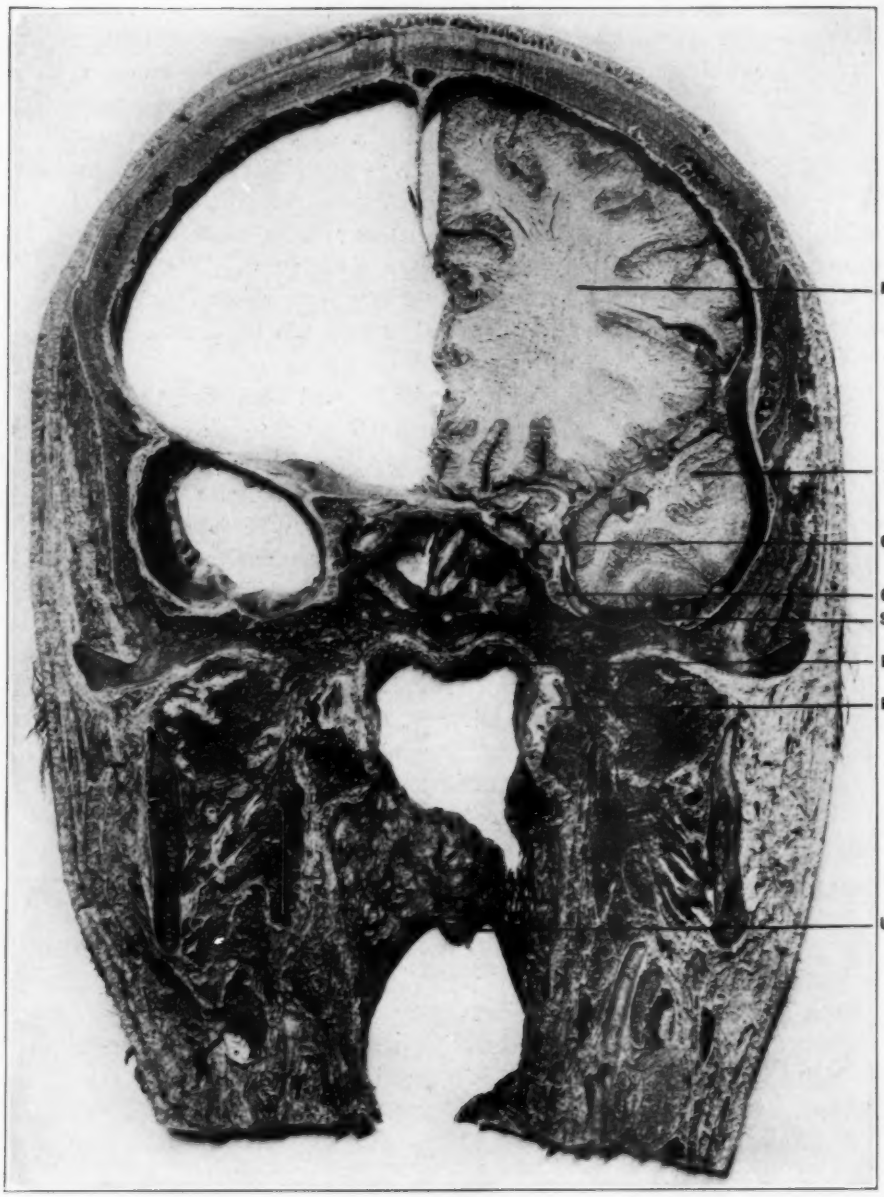

Fig. 19.—Note the cavernous sinus and the trigeminal nerve relationship.

ganglion. All these states may be considered as cranial autonomic, some of nasal origin, others of systemic origin, but influenced by treatment of the nasal ganglion because of the easy approach there to the involuntary

Fig. 20.—Sagittal sections of the head.

nervous system. The interesting feature is that systemic therapy directed toward depression of the cranial autonomic produces a similar effect. In this manner I have supplemented the nasal treatment for the outstanding cranial autonomic conditions with desiccated pituitary whole

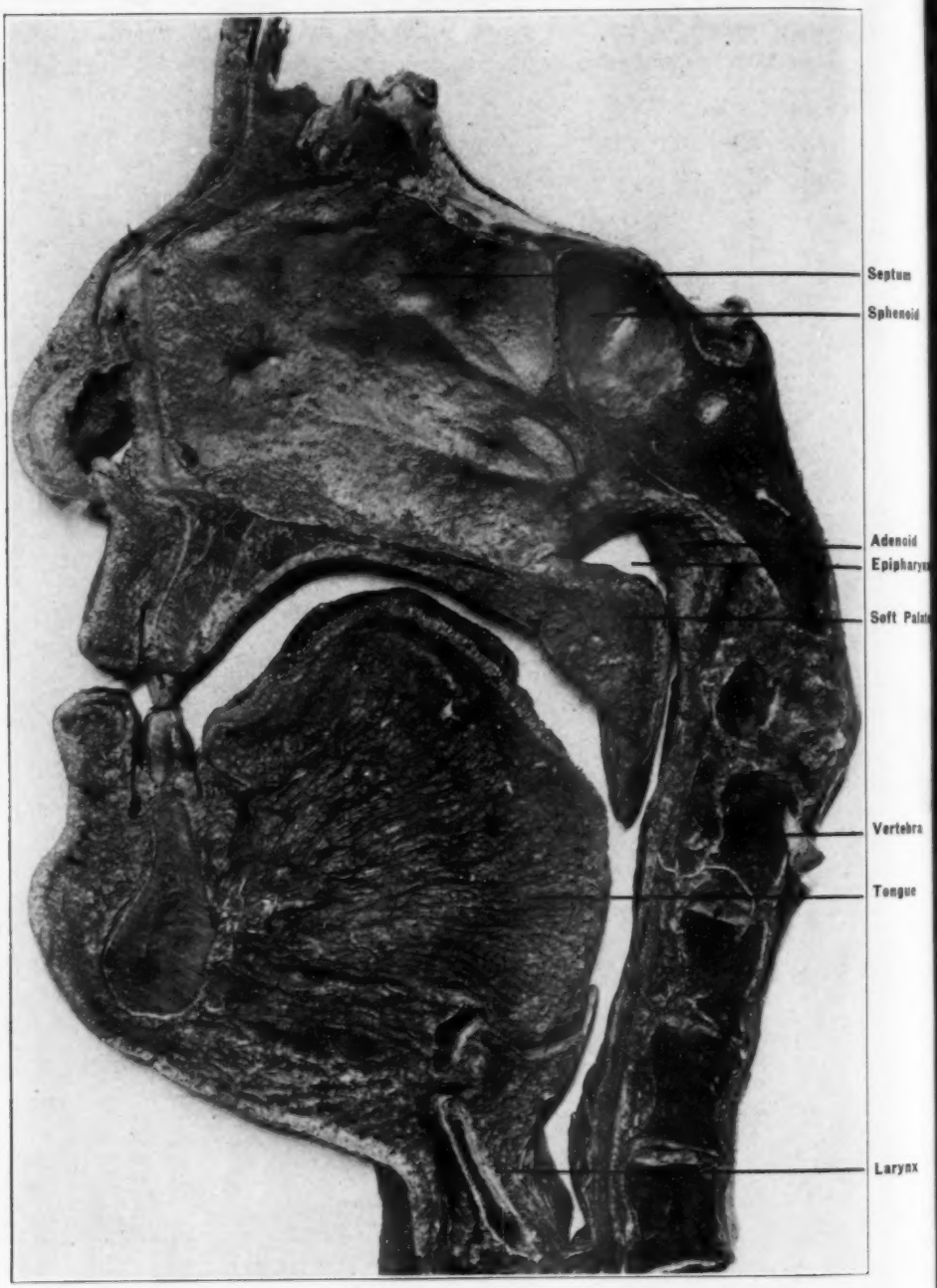

Fig. 21.—Note the pressure markings of the turbinates on the nasal septum.

gland and calcium glycerophosphate. Empirically, these measures have helped in some cases.

Reverting to the facial nerve and the autonomic fibers with it, I believe that some cases of noises in the ear and impaired hearing can be attributed to both vasomotor disturbances in the ear and myalgic conditions of the stapedius muscle. In one of my earlier publications I demonstrated that tinnitus, as well as itching in the ear, can be quickly relieved by cocainization of the nasal ganglion. Winslow likewise claimed good results in tinnitus and impaired hearing by treatment of the nasal ganglion. The itching is also, to my mind, a vasomotor question. In itching of the ear in climacteric cases the administration of a desiccated ovarian preparation has given the same relief as treatment of the nasal ganglion. The sensation of itching of the face is frequently stopped promptly. Herpes, although classically associated with inflammation of the sensory nerves, is apparently also related to vasomotor disturbance of vegetative origin. It would otherwise be difficult to understand the relief from herpes zoster intercostalis (shingles) which I have reported relieved by treatment of the nasal ganglion. I now have a patient with herpes affecting the ophthalmic region of the right side who, after six months of pain, was relieved almost instantly by treatment of the nasal ganglion.

INFLUENCE OF CHRONIC IRRITATION ON THE VEGETATIVE NERVOUS SYSTEM

I come now to the consideration of the third phase of the subject, the influence of chronic irritation on the vegetative nervous system. Fundamentally, in the vegetative nervous system there is a mechanism of balance between two opposing nerve elements; one the cranial and sacral autonomic fibers called by Langley "parasympathetic" coming from the midbrain, medulla and sacral regions, the other "sympathetic" coming from the thoracic and lumbar regions. The former, which I shall refer to as "the craniosacral autonomic," tends toward maintaining an anabolic state of the metabolism through the improvement of digestion and excretion of wastes, the latter, called the sympathetic, tending toward producing a catabolic state of metabolism, in which the body energy and reserves are expended. There are, no doubt, many factors at play in determining the direction of this balance, whether it is toward an anabolic or a catabolic state. I have observed that nasal pathologic changes play an important part in determining the direction of the equilibrium toward catabolism, but also through the direct stimulation of the sympathetic elements coming to the nasal ganglion by way of the deep petrosal as the filaments from around the internal carotid and superior cervical sympathetic. On the other hand, exhaustion of the sympathetic, allowing overaction of the cranial autonomic or direct

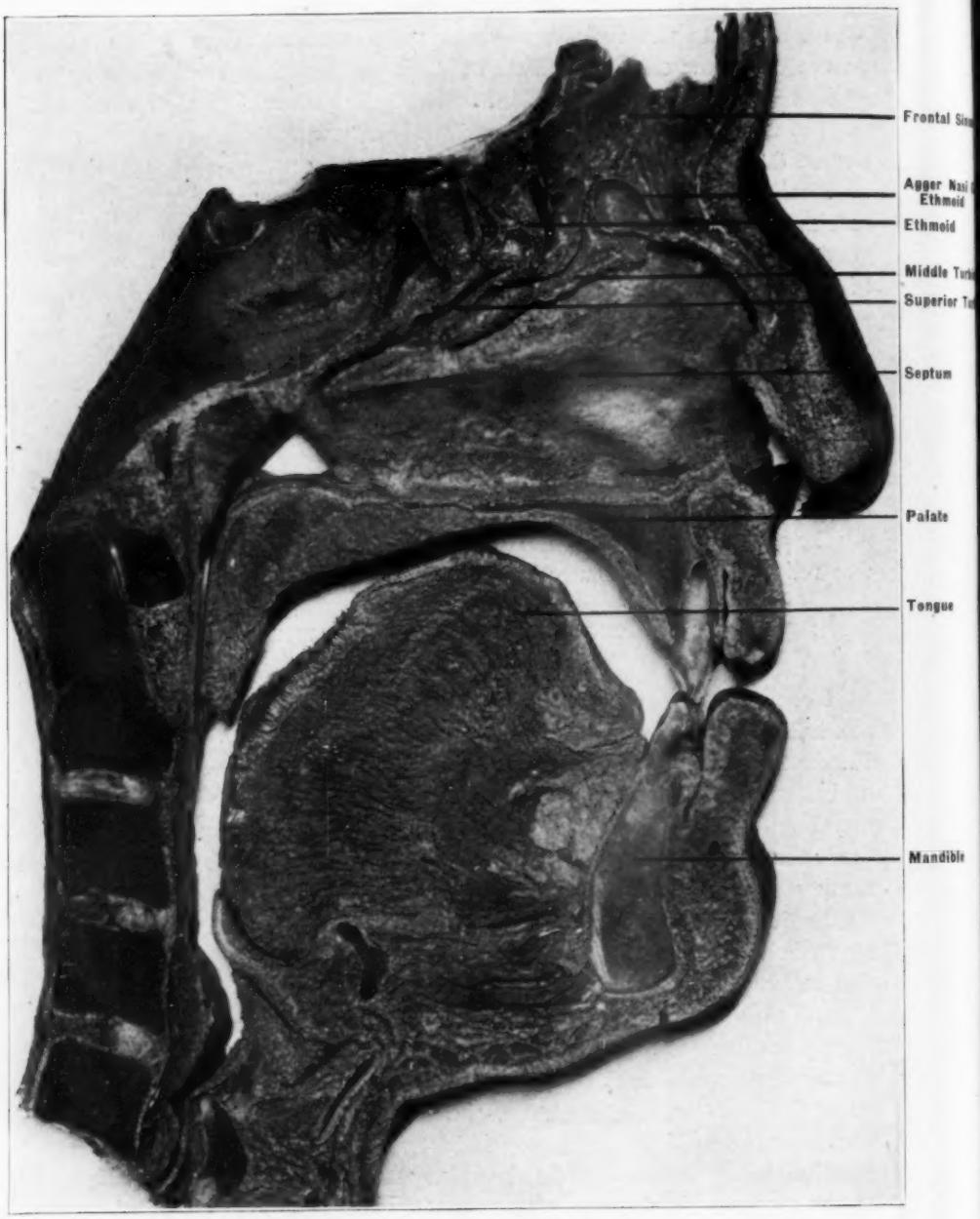

Fig. 22.—Note the sphenopalatine district.

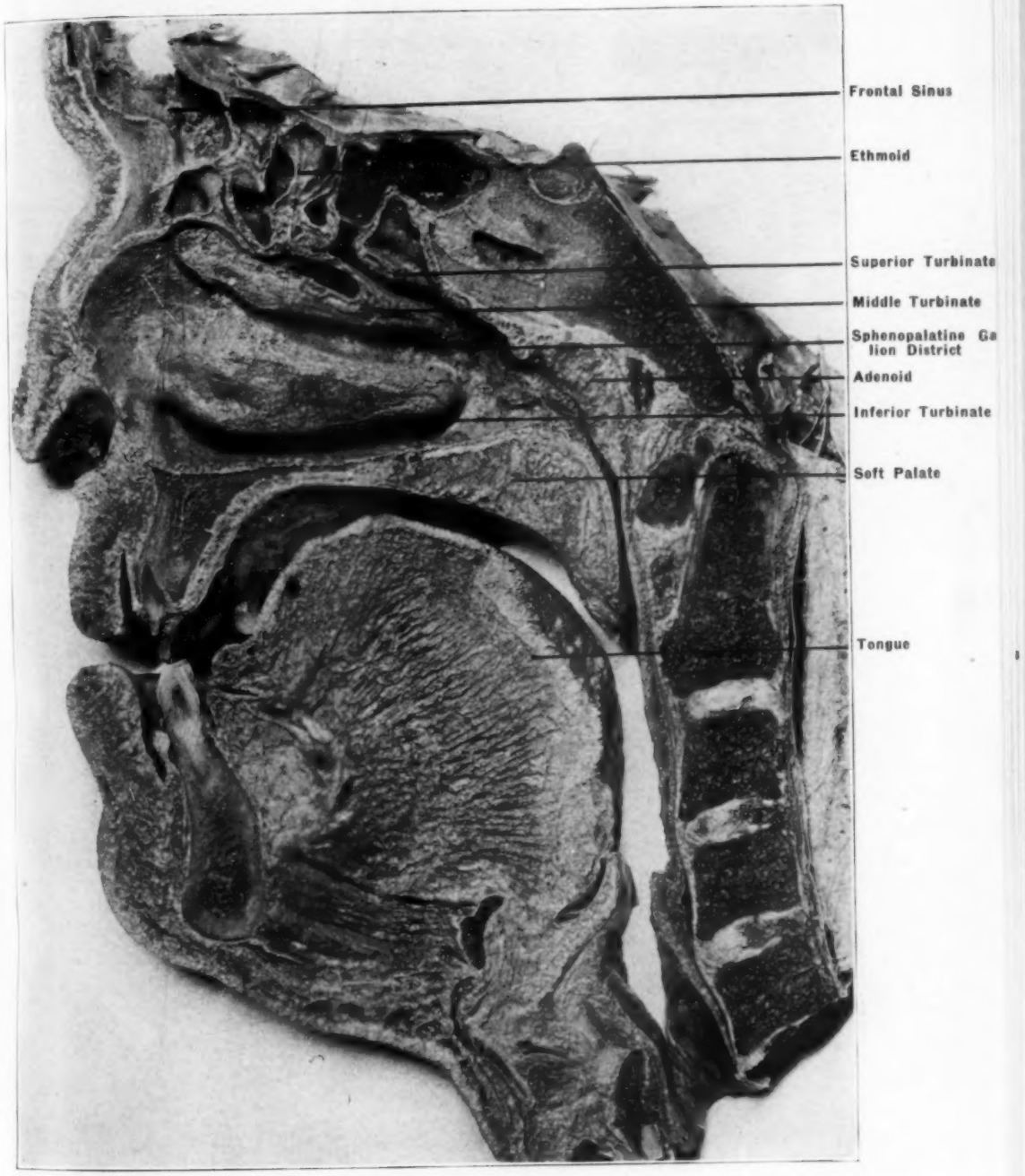

Fig. 23.—Observe the relationship of the turbinates to the nasal ganglion district.

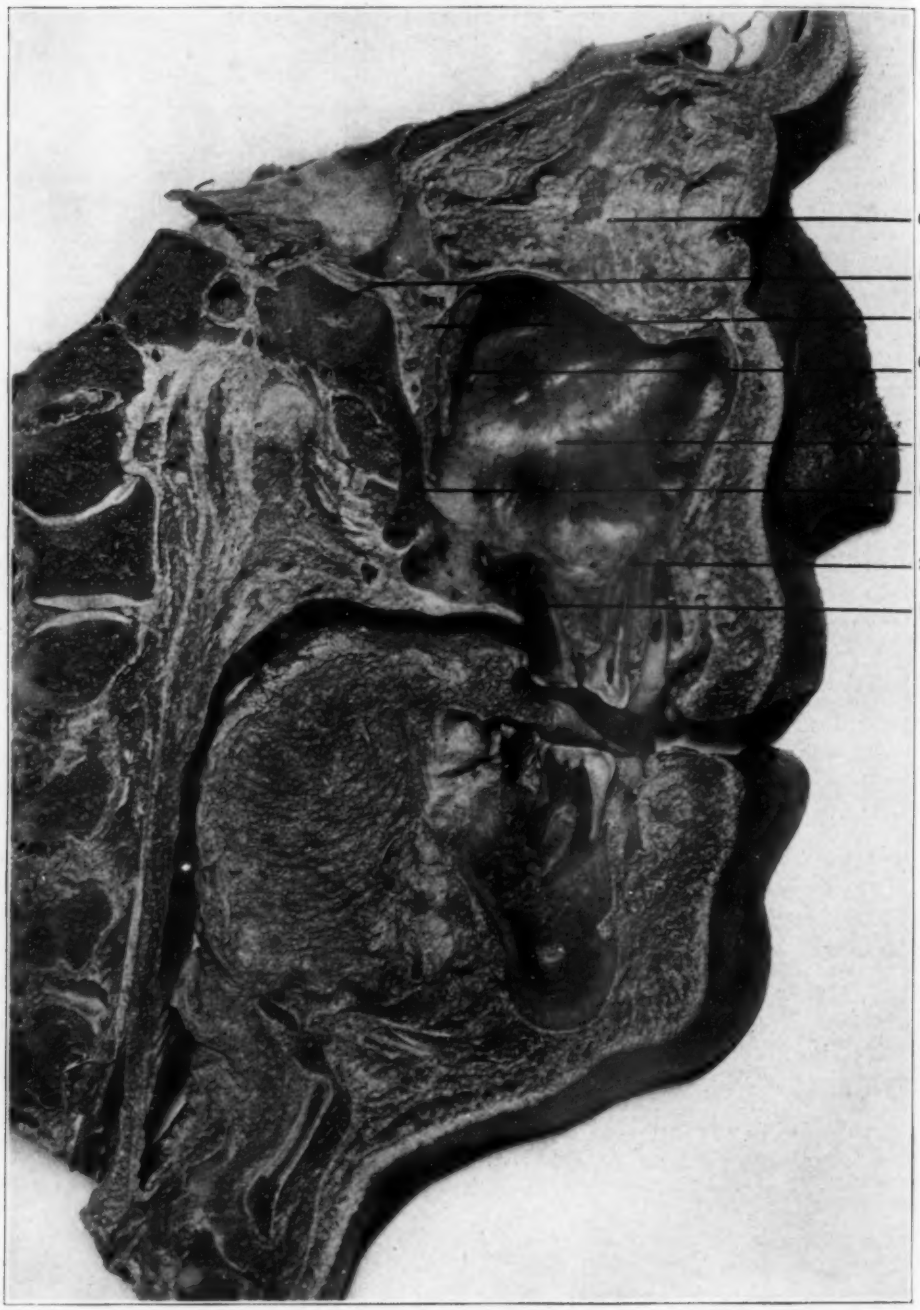

Fig. 24.—Note the chronic inflammatory process in the antrum. No free fluid was found. Typical source of neuralgia.

stimulation of the autonomic in the nose, can throw the metabolism in the direction of the heavy anabolic type. This observation impressed me so emphatically that I now classify most of my cases as anabolic or catabolic, and this gives me a good mental picture of the type of patient I am dealing with. It would, perhaps, be best to illustrate both types by giving a case history of each. Both patients are typical of many of those one is called on to treat. The first case is the catabolic variety:

CASE 1.—Miss D., aged 29, was fairly tall, thin and active, but asthenic looking, long necked, long chested, with underdeveloped breasts, and distinctly underweight. Ten years ago she began to complain of headaches, frontal and occipital, associated with a burning sensation in the eyes, heaviness of the eyelids, heaviness at the top of the head, pains extending over both mastoid regions and pain in the left maxillary region. Six years ago she suffered from an acute attack of Bell's palsy, which lasted for two months and then cleared up entirely. Since the onset of this second attack of pains in the head, she had suffered from tachycardia and nausea, which accompanied each attack. She had received a variety of treatments for the tachycardia, but to no avail. At the time of the examination, she complained of the same pains in the head, palpitation, asthenia and dysmenorrhea. Her history revealed that she had had an attack of acute appendicitis.

Nasal examination revealed a deep red, rather dry mucous membrane, with evidence of a distinct postethmoidal suppuration. She had a septal deflection, impacting on the right, and had had diseased tonsils removed; the larynx was normal. The blood pressure was 102 systolic and 60 diastolic, the pulse rate ranged between 90 and 100, and during the attacks between 100 and 120. I treated the posterior ethmoiditis and administered local applications to the nasal ganglion. Following the treatments she gained 8 pounds (3.6 Kg.), the headaches markedly diminished, the palpitation disappeared and the pains in the head were considerably lessened.

The patients whom I have pictured as suffering from overaction of the sympathetic system from nasal pathologic conditions are extremely sensitive to cocaine, which apparently depresses the cranial autonomic nerve; a small dose brings on a pronounced epinephrine type of reaction, general nervousness, sometimes with weeping, tremor, dilatation of the pupil, pallor and sometimes nausea and vomiting. One must, therefore, also utilize this point in the consideration of anesthesia for operation. In such cases, I usually resort to alypin solution. Frequently, this type of patient will complain chiefly of dysmenorrhea, and it is remarkable to observe how quickly the pain and nausea can be relieved by applications to the nasal ganglion. The older method of destroying the nasal mucous membrane by cautery probably produced its effects by counterirritation of the nasal ganglion. Another effect frequently observed in this type of case is the existence of spastic states in the gastro-intestinal canal, simulating gastric ulcer and cholecystitis. I have in mind, particularly, the case of one patient who had been treated for a gastric ulcer; one gastro-enterologist had told her that she had no ulcer but a nervous stomach. While visiting my office with another patient she had a severe

Fig. 25.—Observe the greater wing of the sphenoid, preventing a needle in the posterior palatine canal from entering the orbit.

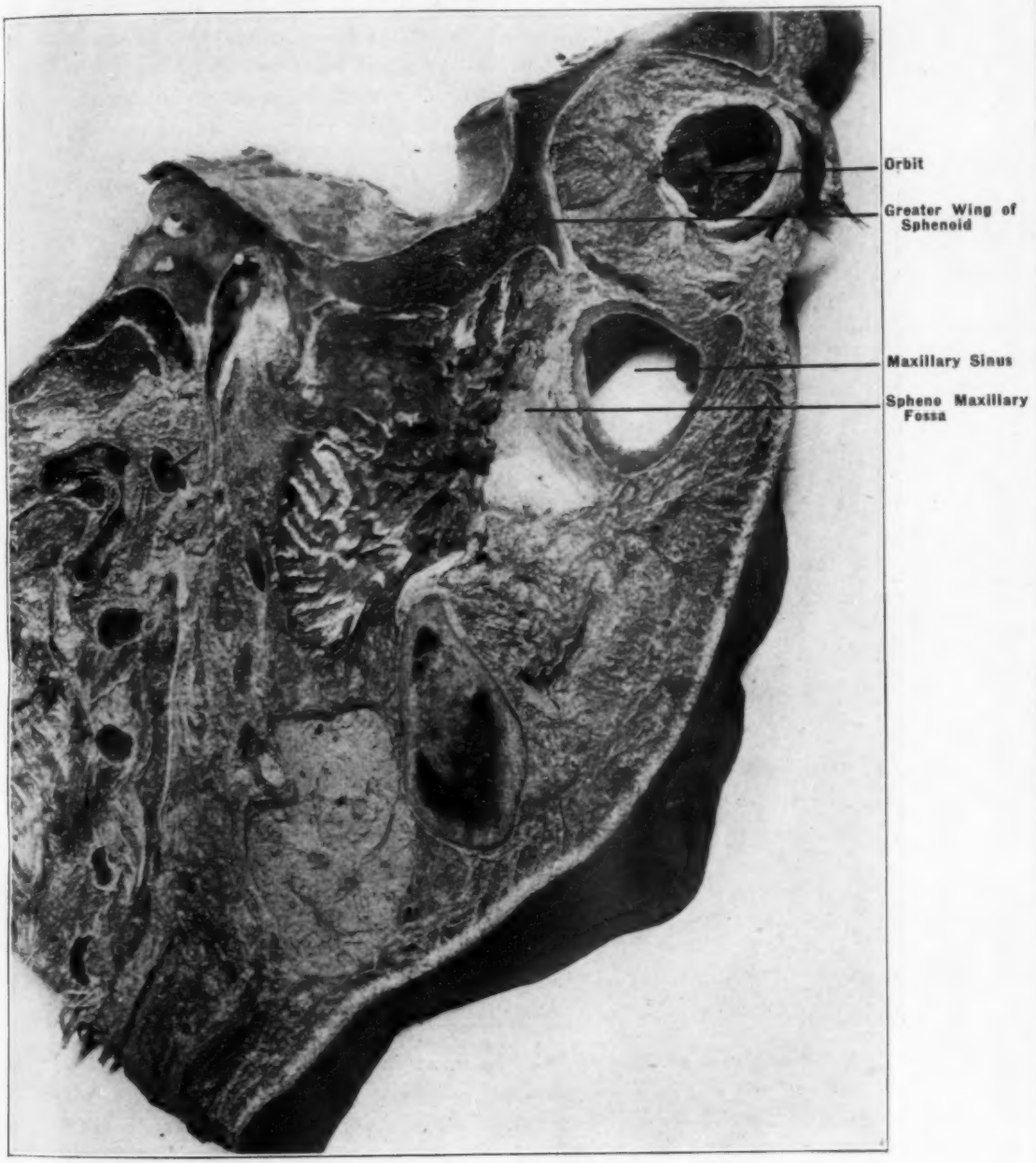

Fig. 26.—Fascia of the sphenomaxillary fossa.

attack of gastric pains. On eliciting her history, I decided to see whether the pain could be blocked by application to the nasal ganglion. I cocainized the nasal ganglion, with instant relief from the pain. She told me that food of any sort induced the attacks and that for the last few months she had been on a practically starvation diet. Following the application to the ganglion, I gave her lunch which she ate in comfort

Fig. 27.—Dissection to show all the sinuses in relationship to the sphenopalatine ganglion.

for the first time since her illness. In conjunction with her gastroenterologist, I followed the case and gave the patient treatments to the nasal ganglion. During the period of treatments she was remarkably relieved. The pathway here is most likely the vagus which is affected through the nasal ganglion. The influence on the respiratory tract is

also great. I have in mind the case of Miss Laf, who suffered from a bilateral maxillary sinusitis apparently for years. In spite of treatment, severe asthmatic attacks developed. These attacks cleared up promptly after several treatments to the nasal ganglion. In this case I was intercepting the irritations coming from the chronic maxillary sinusitis.

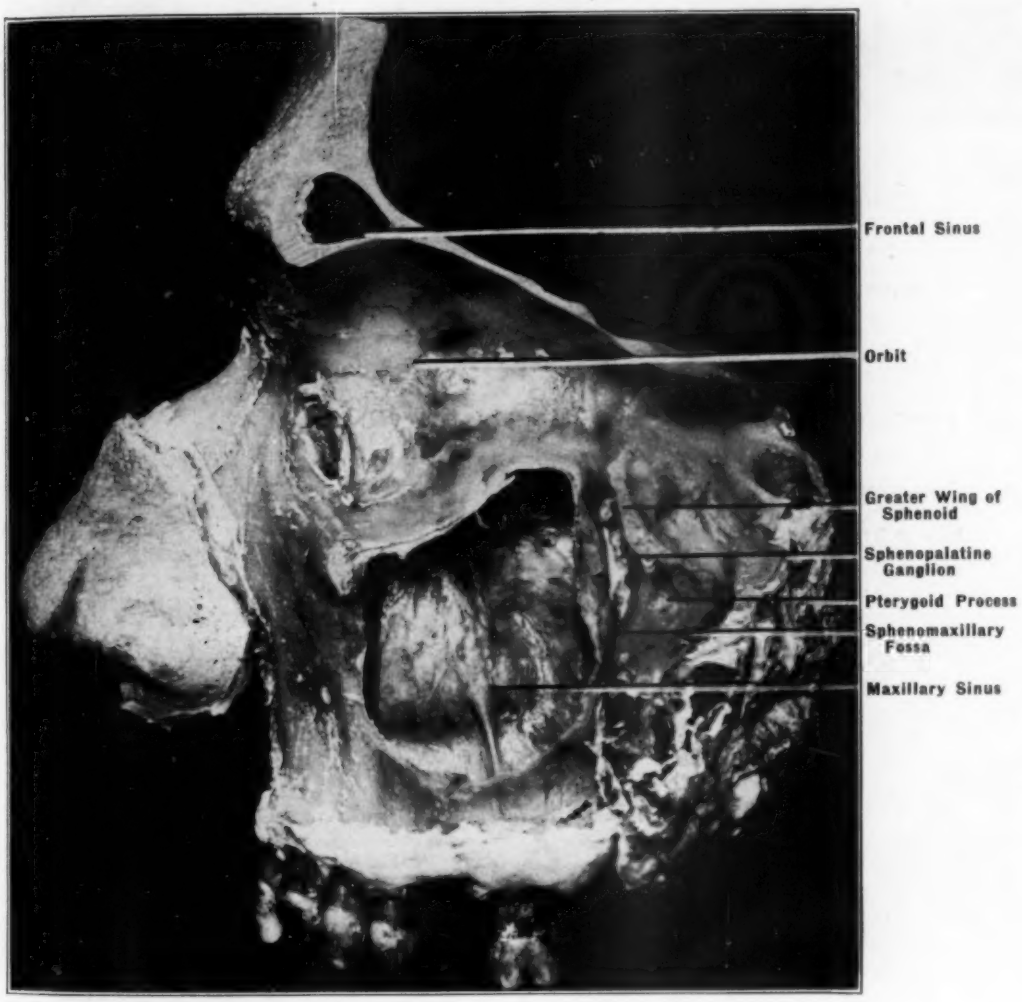

Fig. 28.—Dissection to show the sphenomaxillary fossa.

The mucous membranes of the nose also have a fairly characteristic appearance, and I have here made an observation that will aid materially in the treatment for a common complaint. The mucosa in this type of patient is usually deep red and appears fairly dry. In women one frequently sees the face powder sticking like a paste to the turbinals and

septum. On the floor of the nose one sees a gluelike, tenacious, nonpurulent discharge. Although there is ample space in the nasal fossae the patient will, nevertheless, complain of nasal obstruction and will be continuously puffing through the nose in an effort to break through the veil of mucus with the soft palate. The mucus becomes churned up full of air bubbles and continues to act as an interference to normal respiration. These patients usually travel from one rhinologist to another, complaining of nasal obstruction, and are continuously reassured that

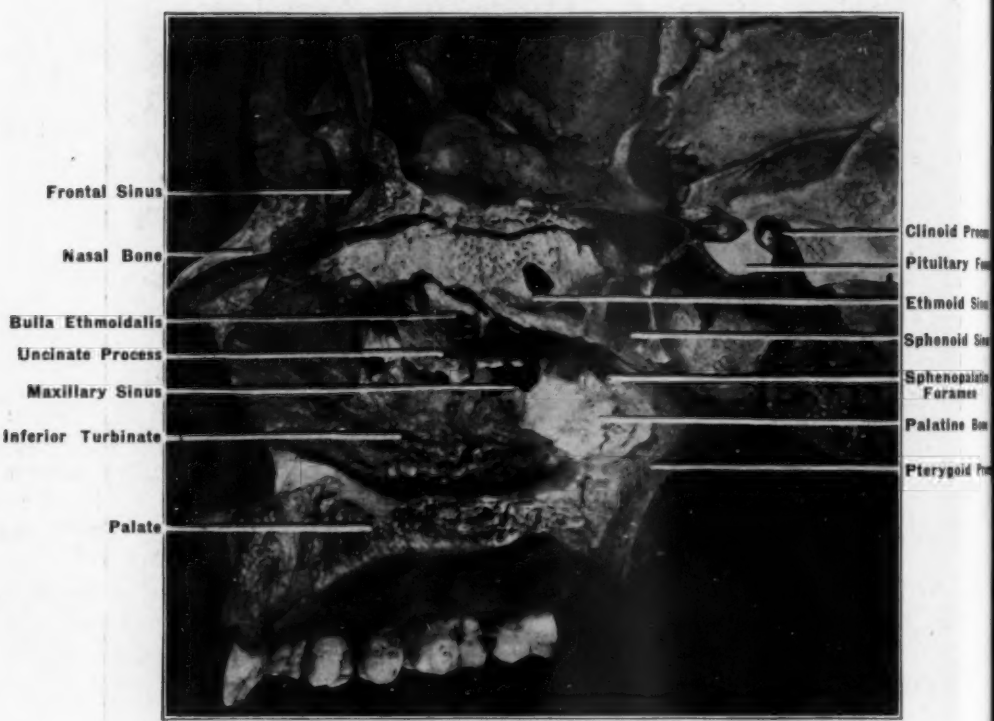

Fig. 29.—Normal relationship of the sphenopalatine foramen.

there is ample breathing space in the nose. I have found that this tenacious mucus is usually formed in the nasopharynx from the mucus-secreting glands, innervated, as Müller has shown, through autonomic fibers to the nasal ganglion. Therapy in these cases must be directed to the epipharynx. I have also seen favorable results in these cases from the use of pilocarpine locally on the nasal ganglion. Attention to this refinement in diagnosis is a great aid in the treatment for the common troublesome complaint of "postnasal dropping" without apparent sinusitis.

In summarizing this catabolic type, I should say that the disturbances are chiefly visceral and spastic while the anabolic type which I am about to describe is chiefly muscular, vasomotor and secretory.

CASE 2.—Mrs. Rose L., aged 44, weighed 195 pounds (88.5 Kg.); she was heavy set and robust looking. For the last twenty years she had been suffering from frontal headaches, radiating to the ear, neck and shoulders—chiefly the left. At the same time she suffered from noninflammatory, so-called rheumatism in the neck, arms and legs so severely as to submit to hospitalization for several months, with only slight improvement. During the last ten years, she had had intensive

Fig. 30.—Note the intimate relationship of the sphenopalatine foramen to the thin walled sphenoid.

endocrine therapy with practically no improvement. During the attacks she suffered with severe nausea, vomiting and vertigo, choking and itching in the ears. She suffered no palpitation or dysmenorrhea. During the time of pregnancy, she had considerable relief. Early in her illness she suffered from an acute gall-bladder attack, for which a cholecystectomy was performed. She had suffered with diseased tonsils, which were removed during her period of hospitalization. Nasal examination revealed a deflected septum, thick hypertrophic mucosa and normal sinuses. Examination of the muscles of the neck and shoulder showed them to be hard, with nodular formation at the origin of the trapezius muscle, especially on the left side. Local application to the nasal ganglion gave immediate

Fig. 31.—Base of the skull.

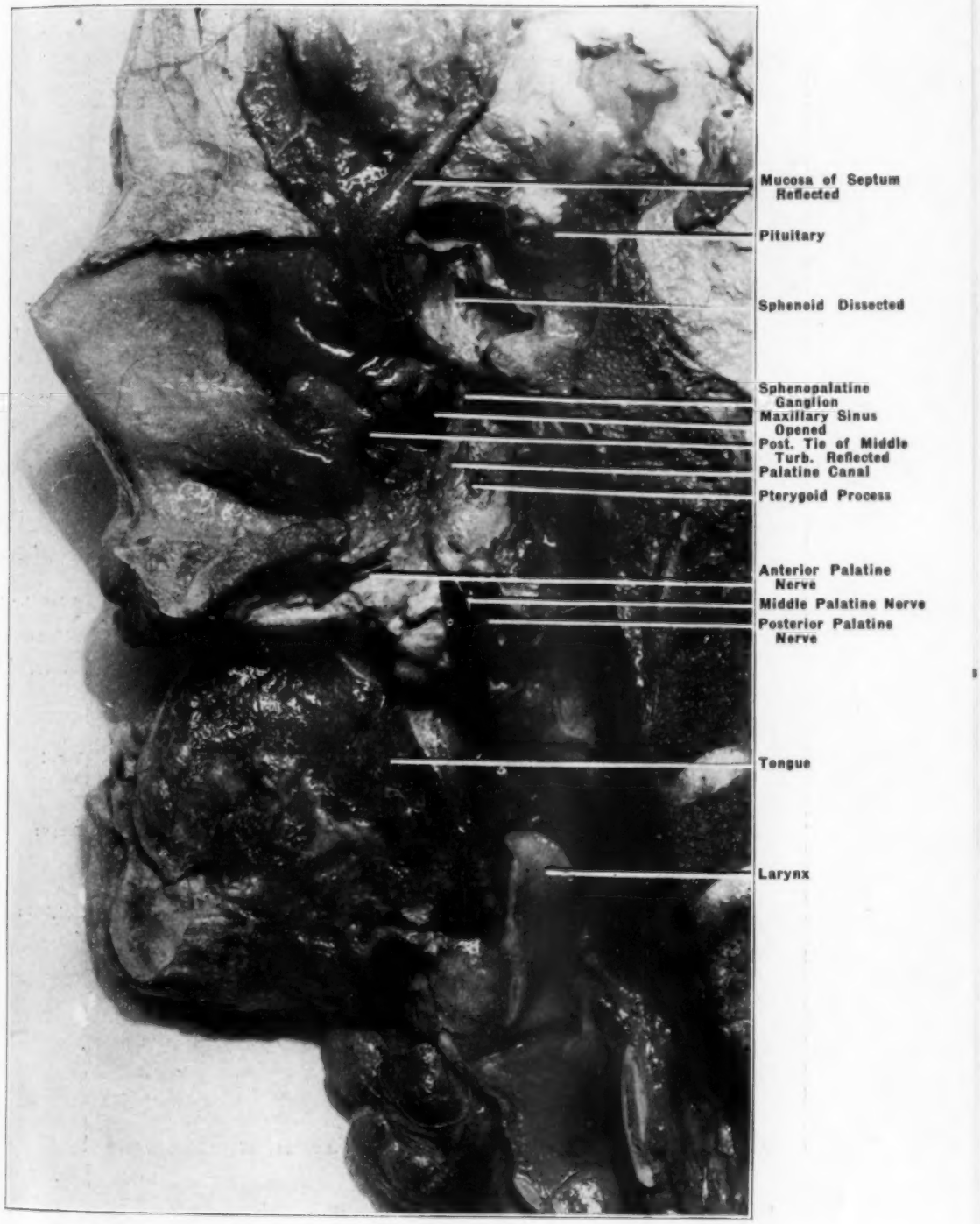

Fig. 32.—Dissection to show the sphenopalatine ganglion (wet specimen).

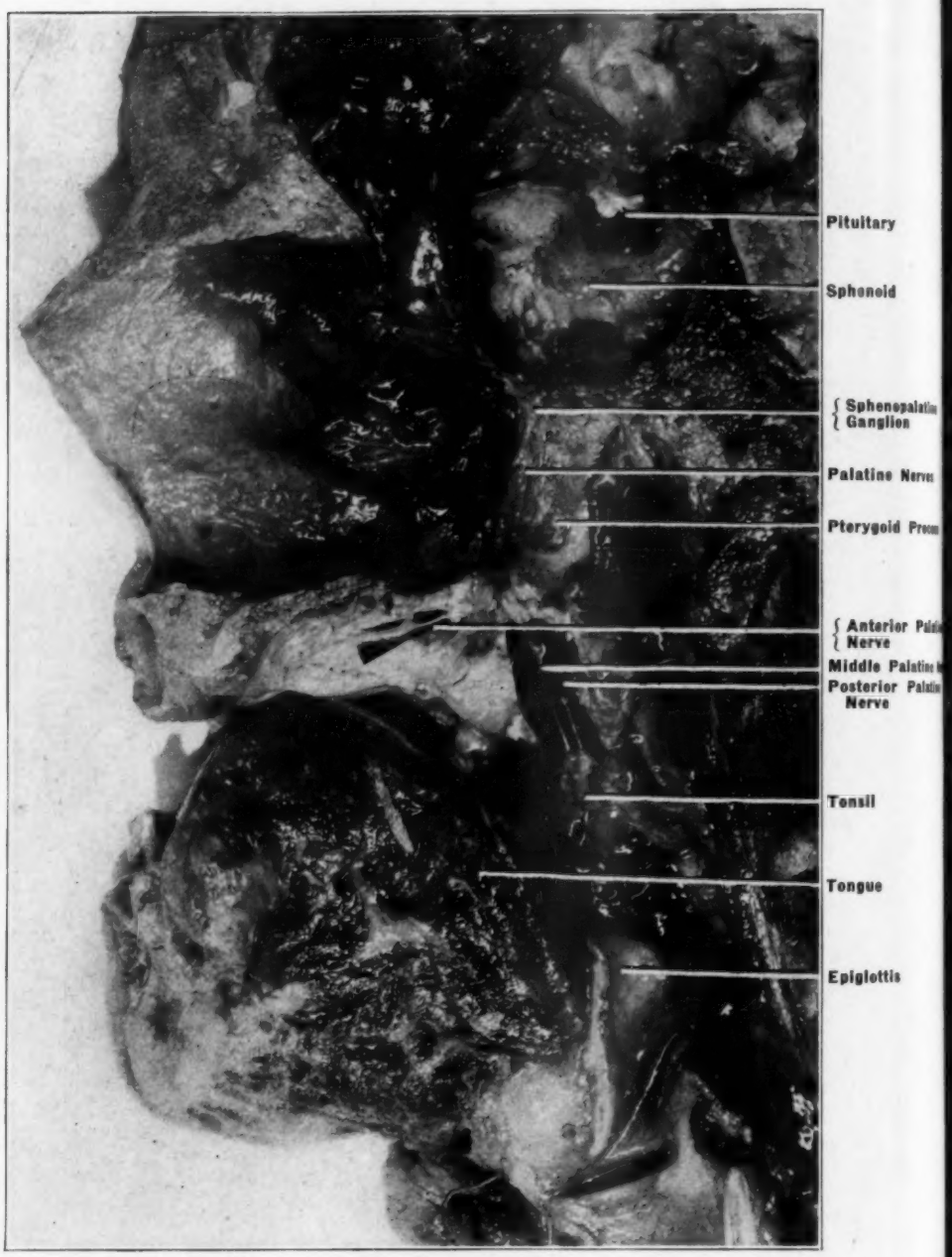

Fig. 33.—Same dissection as in figure 32, to show the middle and posterior palatine nerves supplying the tonsil (wet specimen). Part of the nasal ganglion innervation.

relaxation of these muscles and freedom from the pain. I injected alcohol into the left nasal ganglion and treated the right side conservatively, which resulted in almost complete relief from the headaches and muscular pains.

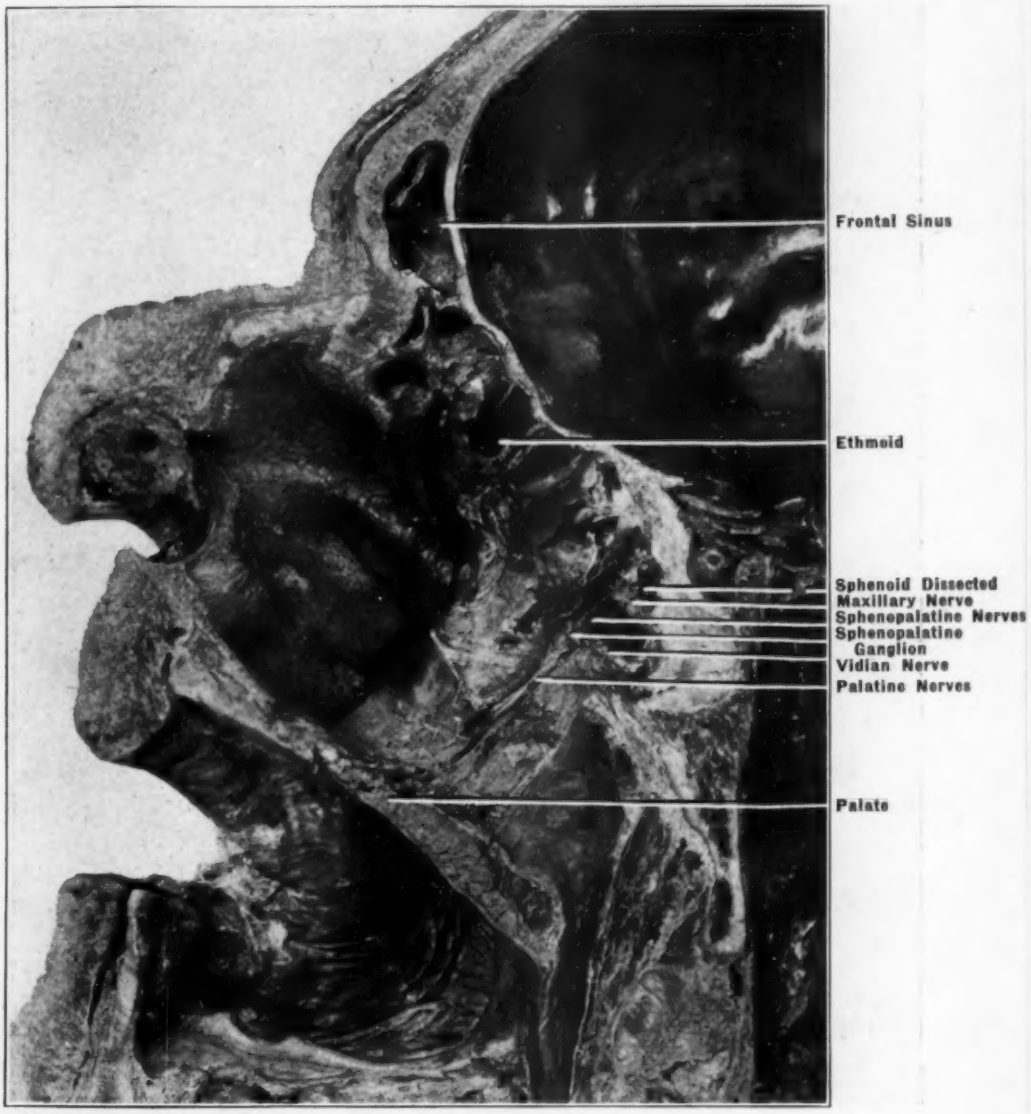

Fig. 34.—Dissection to show the sphenopalatine ganglion.

These cases, to my mind, represent a balance of the vegetative nervous system greatly in favor of the craniosacral autonomic with resultant increased anabolism.

These catabolic and anabolic cases represent the systemic effects of irritation to the nasal nerve. I have still to consider the important question of irritations affecting the local autonomic secretory fibers in the

Fig. 35.—Needle in the posterior palatine canal, showing the approach to the nerve structures.

nasal ganglion. Sneezing and rhinorrhea are the chief symptoms. This sneezing and rhinorrhea can represent either the effect of systemic influences on the nose as in allergic states, or the effect of local nasal

pathologic changes on the nasal ganglion directly. That these vasomotor states react favorably to injection or treatment of the nasal ganglion has long been shown by Sluder, and I have many cases to support his claim. In connection with this type of case, I have made the interesting observation that polypoid ethmoiditis, with or without rhinorrhea, can be markedly improved by the injection of alcohol phenol into the nasal ganglion. Whether this is due to the diminution of the circulation following the fibrosis in the spheno-maxillary fossa, incident to the injection of alcohol, or to the reestablishment of better vasomotor conditions, I cannot say. It suffices, however, to observe that I have been able to circumvent several ethmoid operations in patients who had had previous unsuccessful partial ethmoidectomies by the use of the injections into the nasal ganglion. This represents an interesting and primary use.

THE ENDOCRINE SYSTEM

Before closing, I wish to make some remarks relative to the endocrine system. In a way, it is, perhaps, a storehouse for the vegetative nervous system and acts intimately with it. One cannot help realizing that the pituitary must be consistently influenced by nasal pathologic conditions. Citelli experimentally demonstrated this. Developmentally, the anterior lobe is an epithelial outgrowth from the nasopharynx. It was not, therefore, surprising to me to note that Cushing suggested the existence of cranial autonomic fibers supplying the anterior lobe of the pituitary. Whether this anabolic type which I have described is the result of secondary stimulation of the pituitary by way of the cranial autonomic nerves is merely an interesting possibility in the present stage of knowledge. Whether the type of temporal headache, which now goes under the diagnosis of pituitary headache, is not really one of the cranial autonomics, is a matter for further study. On further observation, I should add that in my studies of sinusitis in children, I find that among the cases of chronic sinusitis proved by the roentgen rays and by positive antrum washings, the majority, contrary to what one would expect, are in heavy set, overweight children with pasty complexions and puffy lower lids. Whether one is here also dealing with stimulation of the cranial autonomic nerves, and indirectly with the pituitary gland, is a matter for future consideration.

SUMMARY

Headaches and systemic disturbances are produced not only by suppurative conditions of the nasal sinuses, but by the irritation of the various nerve tracts long after the suppuration has run its course and stopped.

In these cases the inflammation has left a thickened mucous membrane which acts as a source of irritation to the nerve fibers supplying it.

The roentgen rays show these conditions clearly.

Therapeutically, the cases are nonsurgical and the patients receive but little care.

By treatment of the nasal ganglion the nerve pathway is intercepted between the irritated mucosa and the nervous system, and thus the patient is freed from headaches and systemic disturbances.

Once the nerve pathways are blocked, the thickened sinus membrane is of no further consequence to the patient.

Therefore, there is a rational treatment for the nonsuppurative sinus disturbances.

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VINCENT'S ORGANISMS

IN CHRONIC SINUSITIS, OSTEOMYELITIS OF FRONTAL BONE, ORBITAL CELLULITIS, MENINGITIS AND PULMONARY GANGRENE

REPORT OF A CASE *

DAVID P. SEECOF, M.D.

DENVER

The spirochetes and fusiform bacilli that are commonly grouped as Vincent's organisms are frequently associated with infections in and about the tonsils, teeth, genital organs and gastro-intestinal tract (Davis and Pilot,[1] Varney[2]). The exact rôle played by these organisms in the pathogenesis of the ulcerative and gangrenous process in these and other locations of the body is unknown. According to Lasagna,[3] they "live in symbiosis with other micro-organisms in the necrobiotic ulcer, and together with them determine the initial lesion." Pratt[4] believed that "under certain abnormal conditions these bacteria increase and probably function as secondary infecting agents." Davis, discussing Pearce's paper, "The Specificity of Spirochetes in Diseases of the Eye, Ear and Nose,"[5] stated: "Certain spirochetes are often associated with fusiform bacilli and a pyogenic coccus in their disease activities. These organisms furnish an excellent example of how bacteria may cooperate in this way causing serious infection. Any one of these organisms operating alone would do little or no harm." Moreover, one gathers from the publications of Tunnicliff,[6] Mellon,[7] Varney,[2] Lasagna,[3] Lar-

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* From the Department of Pathology, University of Colorado School of Medicine.

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6. Tunnicliff, Ruth: A Spirochete Associated with Infections of the Accessory Sinuses, J. Infect. Dis. **13**:280, 1913; An Anaerobic Organism Associated with Acute Rhinitis, ibid. **13**:283, 1913; Further Observations on the Bacteriology of Rhinitis with Special Reference to an Anaerobic Organism (B. Rhinitis), ibid. **16**:493, 1915.

7. Mellon, R. R.: A Contribution to the Bacteriology of a Fusospirillary Organism, with Special Reference to Its Life History, J. Bact. **4**:505, 1919.

son and Barron,[8] Davis and Pilot,[1] Krumwiede and Pratt[9] and Pratt[4] that it is not as yet definitely known whether the spirochete and fusiform bacilli are different forms of the same organism or distinct bacteria. The latter two investigators maintained that the fusiform bacilli and the spirochete commonly found together are different and distinct organisms. The object of this communication is not to add any positive data on these questions, but to record a case in which the Vincent organisms were found in the accessory nasal sinuses and in the lesions that appeared as fatal complications following the spread of a chronic infection of these sinuses.

As far as I have been able to determine, there are but few reports published of cases in which these organisms were found in the nose or accessory sinuses. Tunnicliff[6] found the fusiform bacillus and spirochetes in cases of acute and chronic rhinitis. Shulman[10] reported a case of Vincent's infection of the nose and mentioned one other case. Lewis,[11] from a study of the micro-organisms present in suppuration of the accessory sinuses of the nose, concluded that their presence is rare. Hajek[12] (p. 93) merely stated that "at times *Bacillus fusiformis* and *Spirochaeta refringens* are also found, although the definite part they play is unknown." Silberschmidt,[13] according to Varney, found Vincent's organisms in empyema of the antrum of Highmore. Brandt[14] reported an instance of ethmoidal abscess caused by these organisms, in which recovery followed operation and the use of iodine and arsphenamine locally. Pilot and Lederer[15] reported two cases: one in a syphilitic patient, with involvement of ethmoidal cells, and one in which the organisms were recovered from the ethmoid sinuses on operation. Larson and Barron's[8] case in which the fusiform bacillus was isolated

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8. Larson, W. P., and Barron, M.: Report of a Case in Which the Fusiform Bacillus Was Isolated from the Blood Stream, J. Infect. Dis. **13**:429, 1913.

9. Krumwiede, C., Jr., and Pratt, J. S.: Fusiform Bacilli: Cultural Characteristics, J. Infect. Dis. **13**:438, 1913.

10. Shulman, H. I.: Vincent's Infection of the Nose, Am. J. Dis. Child. **36**: 352 (Aug.) 1928.

11. Lewis, C. J.: The Micro-Organisms Present in Suppuration of the Accessory Sinuses of the Nose, J. Path. & Bact. **16**:29, 1911.

12. Hajek, M.: Pathology and Treatment of the Inflammatory Diseases of the Nasal Accessory Sinuses, English translation by Heitzer and Hansel, ed. 5, St. Louis, C. V. Mosby Company, 1926.

13. Silberschmidt: Ueber den Befund von spiessformigen Bacillen (Bacillus fusiformis Vincent) und von Spirillen in einem Oberschenkelabscess beim Menschen, Centralbl. f. Bakteriol. **30**:159, 1901; quoted by Varney (footnote 2).

14. Brandt, F. H.: Ethmoidal Abscess Caused by the Bacillus Fusiformis of Plaut-Vincent, Laryngoscope **23**:1136, 1913.

15. Pilot, I., and Lederer, F. L.: Studies on Fusiform Bacillus and Spirochetes: Occurrence in Putrid Ethmoiditis, Ann. Otol. Rhin. & Laryng. **34**:366, 1925.

from the blood stream two days before the fatal termination may be included. Here there was necrosis of the superior maxilla, with destruction of the bone as far as the antrum of Highmore on the left side and extension into the nasal cavities. The soft palate was necrotic, the hard palate was rough and granular and a foul odor was present.

Their presence in the sinuses and in the locations the involvement of which brought about the fatal termination in the case to be reported was suspected because of the peculiar, acrid, foul odor emanating from the lesions, an odor that I had previously encountered in several cases of proved spirochetal infection of the lung or pleural cavities. Although, as Rist [16] pointed out, lesions with fetid odors frequently result from infections due to *Bacillus coli* and *B. proteus vulgaris*, I am inclined to believe along with Brandt [14] that there is a "characteristic odor" present in spirochetal infections, and with Kline [17] that it is "penetratingly foul smelling." Mellon [7] described a "characteristic putrid odor" of infections with *B. fusiformis*, which he found present in all the discharges of a patient whose blood, when cultured, yielded *B. fusiformis*.

REPORT OF A CASE

Clinical History.—In February, 1927, a girl, aged 12, was sent by the school authorities to the outpatient department of the Colorado General Hospital for correction of defects in vision. At that time, she presented errors of refraction, a divergent concomitant squint, a dacryocystitis on the left side, and, on roentgen examination by Dr. J. L. Harvey, "marked clouding of the frontal, ethmoid and maxillary sinuses" (fig. 1). Operation was advised, but no record of such operation having been done is available.

On Jan. 5, 1929, almost two years later, the patient appeared for the second time at the outpatient department, complaining of headache and pain in the left eye. The pain had come on suddenly three days previously and had been severe until that morning, when it had eased up slightly. She now presented the picture of a very sick child. She was vomiting continuously. The temperature was 100.8 F., and the pulse rate 148. The bridge of the nose was edematous. The supra-orbital ridge and the upper lid on the left side were swollen, tender and purplish, and a small, fluctuating, ill defined mass was felt behind the eyeball. The latter was ptosed downward and outward. There was a marked chemosis of the conjunctiva. The pupils were equal and regular and reacted to light.

A diagnosis of orbital cellulitis was made by Dr. Finnoff, and the patient was immediately admitted to the ward. Here a slight rigidity of the neck was made out in addition to the other conditions. The urine contained granular casts, leukocytes and red cells. The white cells in the blood totaled 16,500, and the differential count showed 98 per cent polymorphonuclears. While local treatments were administered, the patient's condition grew steadily worse, and the tempera-

16. Rist, E.: Neue Methoden und neue Ergebnisse im Gebiete der bacteriologischen Untersuchung gangränoser und fötider Eiterungen, Centralbl. f. Bakteriol. (orig.) **30**:287, 1901.

17. Kline, B. S.: Pulmonary Abscess and Pulmonary Gangrene, J. A. M. A. **90**:2008 (June 23) 1928.

ture rose to 105 F. during the night. In the morning, a small incision was made in the left upper lid. A large amount of thick, malodorous pus was obtained. It was reported to contain "gram-positive streptococci and large gram-negative rods" (by Mr. A. L. Scott of the department of bacteriology). The patient soon developed Cheyne-Stokes respiration and died during the afternoon. The necropsy was performed nineteen hours later.

Observations at Necropsy.—The body was well developed, though slightly emaciated, and weighed 82 pounds (37.2 Kg). There were no abnormalities of

Fig. 1.—Roentgenogram showing small cloudy frontal sinuses and opaque antra and ethmoid sinuses.

the scalp, except that in the left frontal region, over an area 10 by 7 cm. in diameter, it was separated for about 2 cm. from the calvarium by a space in which some fluid and gas could be felt. The left upper eyelid was purplish black, and at the supra-orbital ridge there was a recent linear incision about 1 cm. long. There was a small, well healed crucial scar over the medial angle of the supra-orbital ridge on the right side. The pupils were equal, regular, centrally placed and in mid-dilatation. The ears and nose showed no abnormalities externally. The mucous membranes of the mouth were cyanotic. The teeth were well kept, and well preserved. The gums were clean. The organs of the neck were not removed.

The thymus was small. The heart, except for innumerable petechial hemorrhages in the epicardium, was normal.

All three lobes of the right lung were voluminous and soggy. The pleural surfaces throughout were thin, smooth and glistening. The upper two lobes were uniform in consistency. The lower lobe contained scattered areas of consolidation, varying from 1 to 3 cm. in diameter. These showed on the surface as localized,

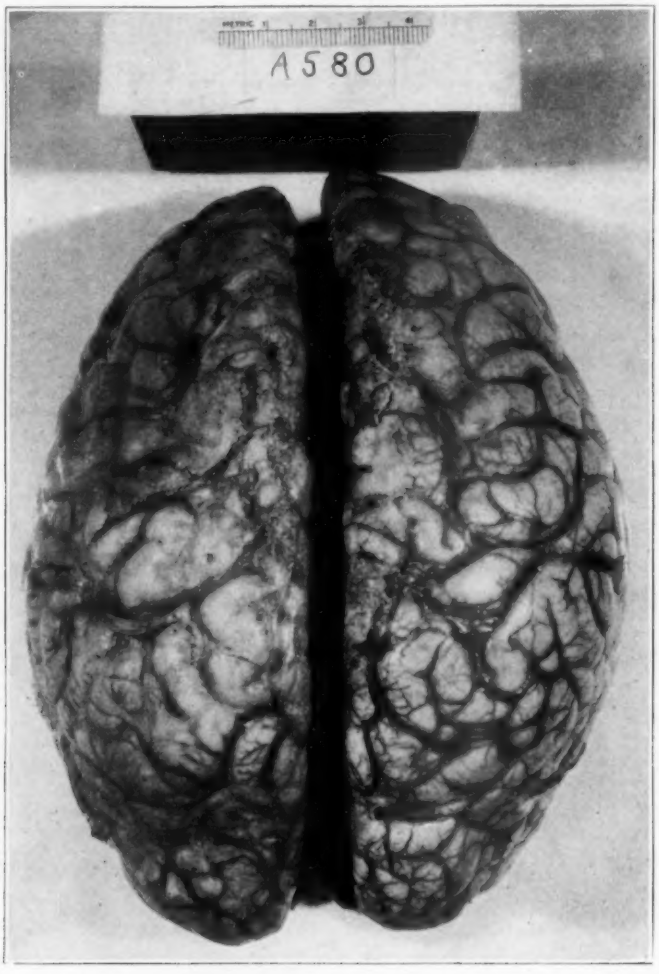

Fig. 2.—Exudate on left hemisphere of brain.

slightly elevated, bluish-black areas. On section, there was a moderate passive congestion, with edema, throughout. The consolidated areas in the lower lobe were irregularly globular in outline; in places, however, they were somewhat wedge-shaped. The consolidated portions were firm, dark red and granular. In a few there were small central areas of grayish-white necrosis. The bronchi were filled with thick mucopurulent exudates. The mucous membranes showed injection. The vessels were slightly thicker than the average. The root glands were

small, anthracotic and marked by injection. The left lung was essentially similar to the right, showing areas of consolidation with central foci of necrosis in the lower lobe, marked passive congestion and edema throughout, and purulent bronchitis.

The liver weighed 1,620 Gm. and except for a marked granular degeneration was normal. The gallbladder and ducts were normal. The spleen weighed 150

Fig. 3.—Base of skull after exposure of sinuses and orbits, showing necrosis of fat in left orbit (*A*), and exudates in frontal (*B*), ethmoid (*C*) and sphenoid (*D*) sinuses.

Gm. and was larger and firmer than normal; the malpighian bodies were prominent.

The gastro-intestinal tract was congested throughout; the stomach contained several small submucous hemorrhages, and the lymphoid tissues of the ileum and colon were moderately enlarged. The appendix was normal. The pancreas was normal.

The suprarenal glands showed slight hypertrophy of the cortices and marked congestion of the medullas; the right weighed 12 Gm. and the left 8 Gm. The kidneys showed marked granular degeneration; the pelves and the ureters showed innumerable petechial hemorrhages. The urinary bladder and the internal genitalia were normal.

When the scalp was stripped off, the fluctuating area over the left fronto-parietal region was found to contain a small amount of hemorrhagic purulent material and gas. This material gave forth a pungent, acrid foul-smelling odor (characteristically that of spirochetal infections). The inner surface of the scalp

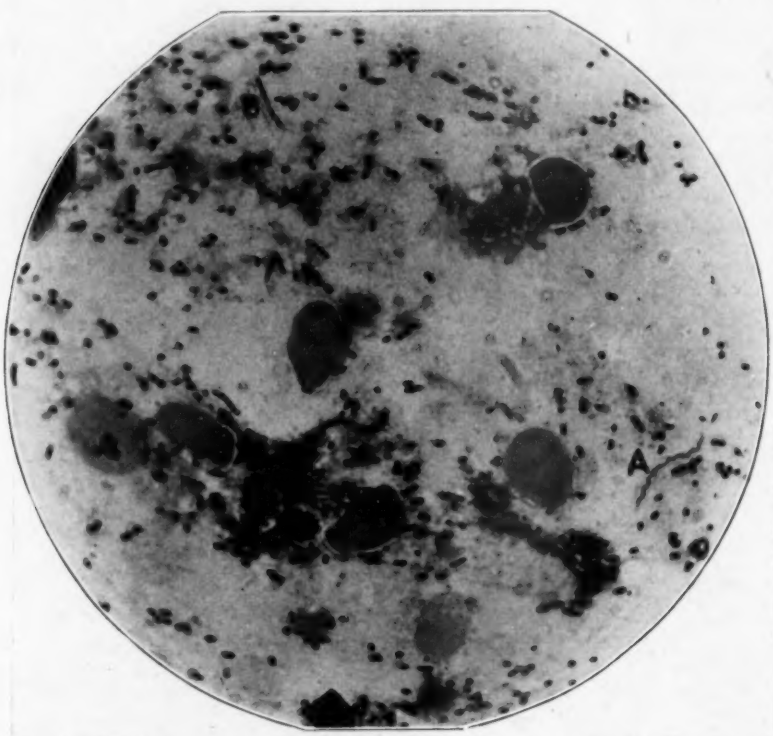

Fig. 4.—Spirochete (*A*), fusiform bacillus (*B*) (granular form) and other organisms in smear from pus; Giemsa stain.

over this area showed slight superficial erosion; the underlying calvarium presented a slight amount of injection, but was not eroded. There was moderate edema of the scalp over the posterior left half; elsewhere the scalp was normal. (There was no communication between the localized area and the incised wound in the supra-orbital region on the left side.) On removal of the calvarium, several areas of greenish purulent exudation were seen on the outer surface of the dura on the left side. These areas varied from 2 to 3 cm. in diameter. There were areas of greenish discoloration on the under surface of the calvarium overlying the discolored areas of the dura (small portion removed). There was no erosion of the calvarium. The right half of the dura appeared normal. On removal of the dura, the entire left hemisphere was found to be covered by a thick layer

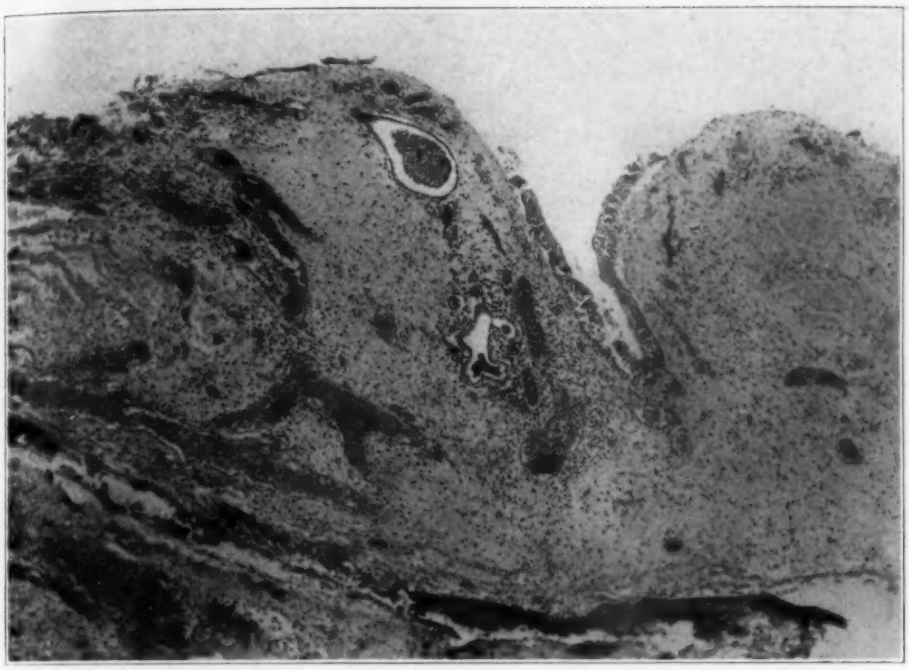

Fig. 5.—Section of ethmoid sinus showing chronic inflammatory reaction of mucous membrane, and at left, necrosis extending down to and including bony lamellae.

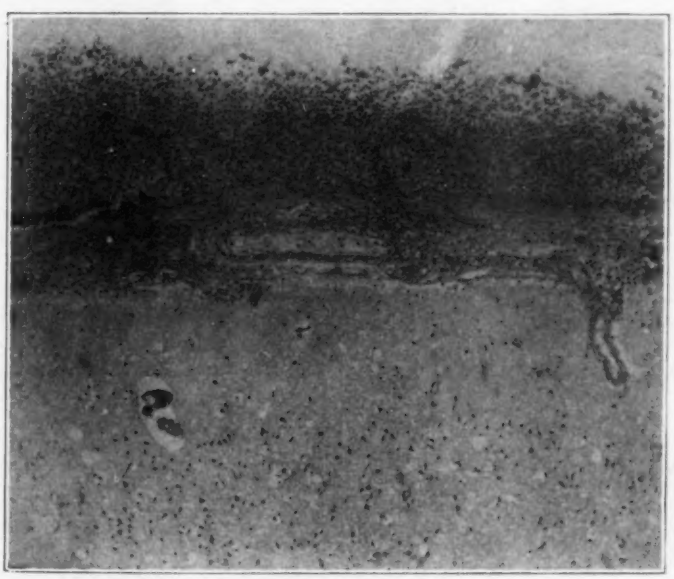

Fig. 6.—Purulent exudate on surface of brain.

Fig. 7.—Cross-section through frontal bone showing purulent and necrotic exudates in diploë and erosion of lamellae. The field mapped out is enlarged in figure 8.

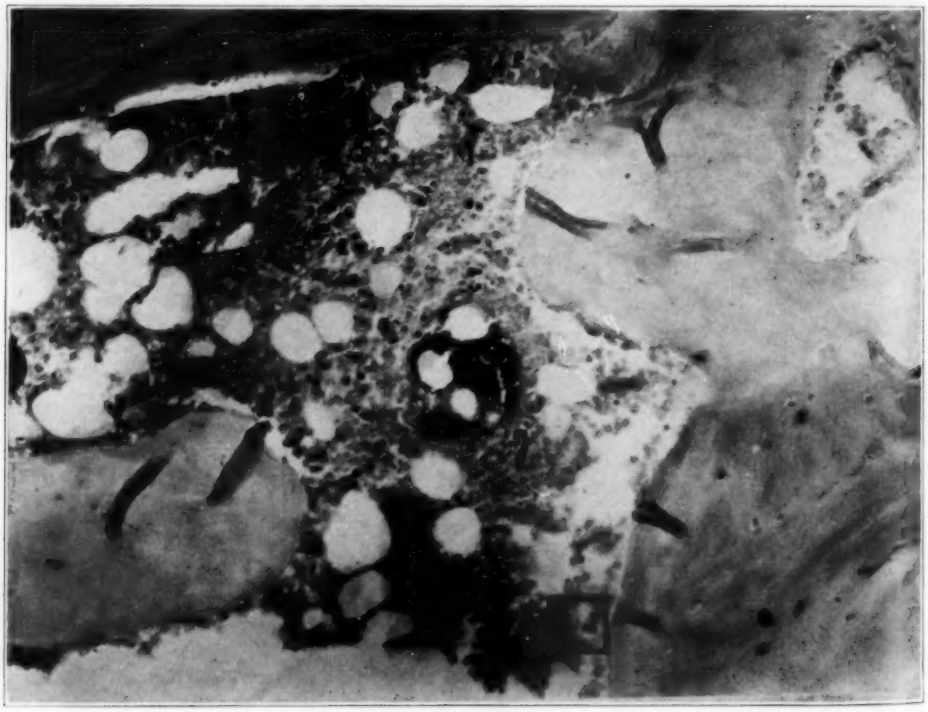

Fig. 8.—Purulent and necrotic exudate, and bacterial masses in diploë. The field mapped out is enlarged in figure 10.

of mucoid, greenish, purulent material giving forth an acrid, pungent odor. The purulent material was strikingly limited to the convexity of the left hemisphere (fig. 2). The meninges covering the right hemisphere were normal. The meninges at the base of the brain were normal.

The brain weighed 1,200 Gm. On section, the ventricles were found to be slightly dilated, but the fluid was clear.

Exploration of the cavernous sinuses revealed no abnormalities. No perforation or erosion of the bones of the base of the skull was made out.

When the sinuses—frontal, ethmoid and sphenoid—were opened, large amounts of mucoid, greenish-brown, acridly foul-smelling material were found filling them

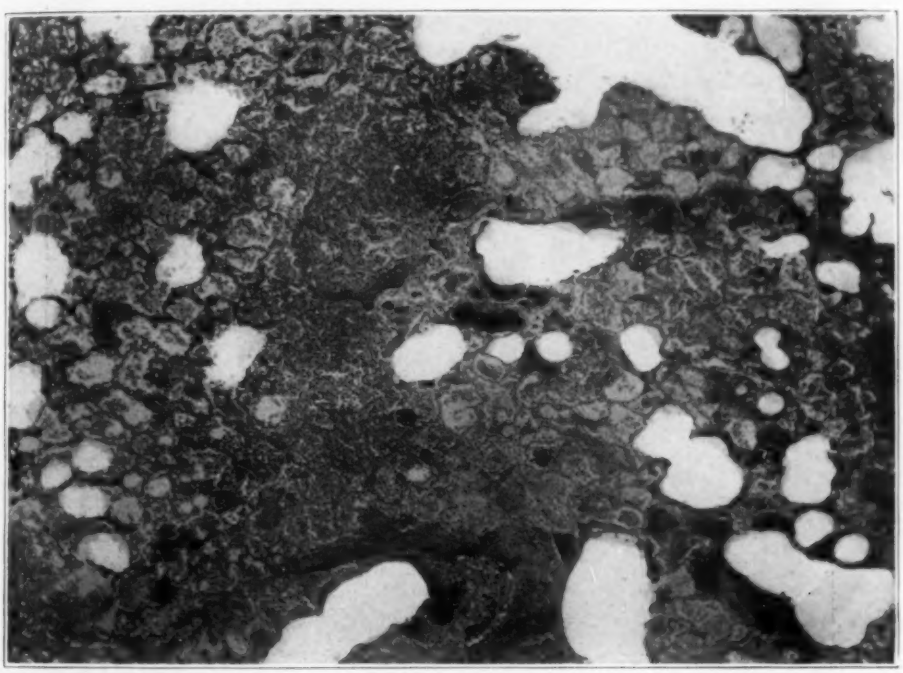

Fig. 9.—Lung showing purulent exudates in alveoli at left, and at right bacterial masses and necrosis of parenchyma.

completely. In the left orbit, the fat was necrotic, and all the contents were bathed in purulent material similar to that found in the sinuses. The orbital contents on the right side were normal. The mastoid bones were normal (fig. 3). The maxillary sinuses were not explored.

In smears from the pus under the scalp, and from the frontal, ethmoid and sphenoid sinuses, left orbit and dura, stained with methylene blue (methylthionine chloride, U. S. P.), fuchsin, and Gram's, Fontana's and Giemsa's solutions, I found, in addition to staphylococci, streptococci and large, thick, gram-negative rods, innumerable spirochetes and fusiform bacilli of the Vincent types (fig. 4).

Cultures from the heart blood showed "*B. coli* and *Streptococcus fecalis*," and those from the pus of the sinuses, orbit, dura and scalp, "*B. coli* and unidentified gram-positive nonpathogenic *Lactobacillus*" (A. L. Scott).

Histologic Observations.—The ethmoid tissues revealed edema, scattered areas of round cell infiltration, chiefly perivascular, and foci of purulent necrosis extending down to and including the bony lamellae (fig. 5).

The brain showed a fibrinopurulent exudation in the pia and on the free surface (fig. 6). There was a fibrinopurulent layer on the inner surface of the dura, and at one place an area of purulent necrosis involving the entire thickness. The calvarium (decalcified) had the outer tables intact. Necrotizing, purulent exudates replaced the marrow, distending the bony lacunae and eroding the adjacent surfaces of the bony lamellae (figs. 7 and 8).

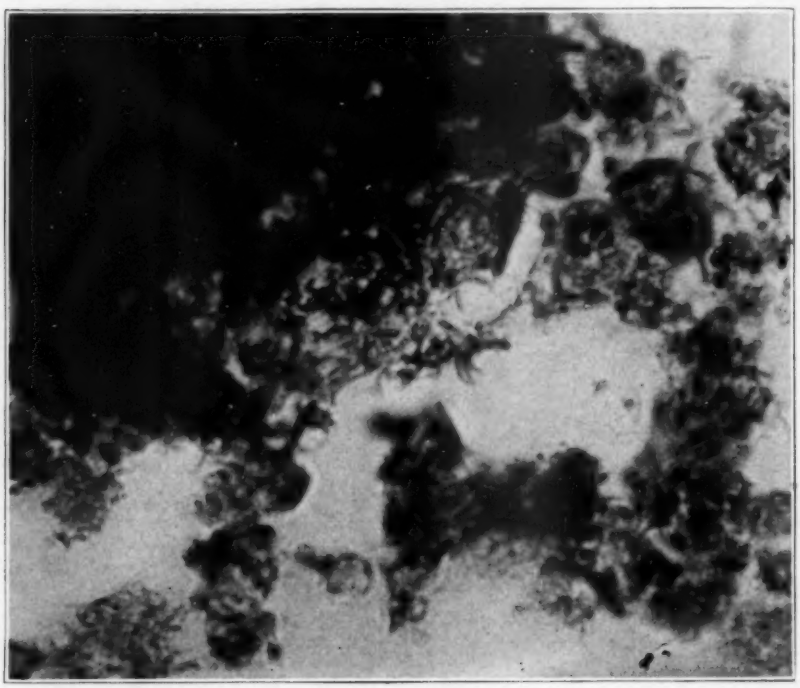

Fig. 10.—Fusiform bacilli in osteomyelitis of frontal bone. Oil immersion of field marked out in figure 8.

The lungs (numerous sections) revealed congestion and edema, acute bronchitis, foci of hemorrhagic bronchopneumonia and scattered areas of recent infarction and anemic necrosis (gangrene) (fig. 9).

In the other organs the important observations were: congestion and foci of free hemorrhage in the heart, liver, stomach, kidneys and suprarenal glands, and hypertrophy of the lymphoid foci in the intestines.

Bacteriologic Observations.—Gram's stain, methylene blue, fuchsin and Giemsa's and Levaditi's stains were used for all the organs removed. In addition to staphylococci, streptococci and rods of various sizes and staining, the spirochetes and fusiform bacilli of the types grouped as Vincent's organisms were found in the purulent and necrotic foci in the sections of the ethmoid bone, dura, cerebral meninges, calvarium (frontal bone) and lung. The spirochetes were more

numerous than the fusiform bacilli in the lung; but were present in small numbers in the calvarium, where the latter predominated (fig. 10). In the remainder of the organs mentioned, both forms occurred in practically equal numbers. No organisms were found in any of the other organs.

COMMENT

Identification of Vincent's Organisms.—In numerous recorded bacteriologic studies of the contents of the sinuses and of the secondary foci of involvement no mention is made of anaerobic cultures. The opinion is ventured that in many instances of fetid conditions, these organisms may have been missed. Indeed, in the case reported, their presence was established only after a deliberate search for them had been made in the smears and tissues; and the failure of others to grow them may be ascribed to the fact that their presence was not suspected, and appropriate culture mediums were not employed. Lasagna [3] pointed out the difficulty of isolating them in the presence of the symbiotic organisms, especially the pneumococcus, streptococcus and staphylococcus. For practical purposes, the suggestion offered by Rist [16] may be useful. He advised that, in doubtful cases, smears be made at once, and if these anaerobic organisms are seen, suitable methods and culture mediums can then be successfully employed. It is difficult, however, to suggest any satisfactory mediums for the routine cultivation of Vincent's organisms. The detection of these organisms, I believe, may have an important bearing on the therapy (see a following paragraph on therapy), and in the final analysis, their detection depends on a careful examination of the smears in suspected cases, especially those in which the "characteristic odor" suggests their presence.

Localization of the Organisms.—Pratt [4] established that fusiform bacilli are present normally in small numbers in the throat, having found them in 100 per cent of the men and monkeys, in 60 per cent of the guinea-pigs and in 48 per cent of the rabbits of the series examined. Their presence in carious teeth, on the gums and in the tonsils is well known. In the case reported, the teeth, gums and tonsils were, grossly, normal. In this respect, the observations resemble those in one of the two cases reported by Pilot and Lederer,[15] in which the teeth were good and the tonsils had been removed.

It is possible that the small numbers present in the mouth and throat found their way into the sinuses, where, because of the chronic inflammatory reaction (with the resulting failure of the ciliary action and bactericidal action of the secretions), and, probably, because of the shutting off of the circulation of air with the establishment of "anaerobic" conditions, these organisms found ideal conditions for growth. Rist [16] accounted for their occurrence in otitic inflammations on the ground of the anaerobic conditions present, which favor their growth after they have reached these locations via the eustachian tube.

The presence of the organisms in the purulent exudate on the meninges in association with a spread from otitic foci is not a rare observation. Ghon and his associates [18] recorded one case (case 3), and cited Rist as having, in 1898, described such an occurrence along with multiple gangrenous pulmonary infarcts, as in my case.

As far as I have been able to determine, this is the first instance in which Vincent's organisms have been found in osteomyelitis of the cranial bones secondary to sinus involvement, with the possible exception of one of the two cases reported by Pilot and Lederer,[15] in which "in Levaditi preparations of the decalcified bone (of the ethmoid), many fusiform bacilli, spirochetes and cocci occurred in the exudates, but no spirochetes could be seen in the bony tissue."

Moreover, their presence in the diploë of the frontal bone indicates the mode of extension of the process in this case. Freudenthal [19] stated that the "venous sinuses of the cranium, as well as the intracranial veins, and those of the diploë have no valves, and, therefore, the thrombus is easily carried along." Brandt [20] in reporting a case of meningitis, etc., following frontal sinusitis (not spirochetal) explained its occurrence as follows: "A violent infection produced the osteomyelitis, and by contiguity the brain abscess, and later by further extension of the bone process, caused the meningitis." In the absence of visible foci of perforation at the base of the skull, it must be concluded that the extension took place "along veins into the diploë, and out through both surfaces via the venae perforantes" giving the abscess under the scalp on the external, and the meningitis on the internal, surfaces of the frontal bone.

It may be mentioned, also, that the presence of the osteomyelitis of the frontal bone in this case was detected "accidentally" on histologic examination. In the gross, except for the discoloration of the surface, the bone appeared normal. I was not familiar with the facts, as pointed out by several authors, that "macroscopically there may be no destruction of the bone, yet isolated stained areas of grayish-red or bluish-gray may be seen in the bone," these areas corresponding "to the perforating veins" (Hajek, p. 118); and that "in other cases the bone was found macroscopically intact, whilst the dura mater over it was inflamed and microscopic changes were present in the bone" (Moritz,[21] in a review of seventy-six cases with cerebral complications).

18. Ghon, A.; Mucha, V., and Müller, R.: Beiträge zur Kenntnis der anäeroben Bacterien des Menschen: IV. Zur Aetiologie der akuten Meningitis, Centralbl. f. Bakteriol. (orig.) **41**:305, 1906.

19. Freudenthal, W.: Endocranial Complications of Nasal Origin, Laryngoscope **20**:60, 1910.

20. Brandt, F. H.: Brain Abscess in Frontal Lobe Resulting from Acute Frontal Sinusitis and Osteomyelitis of Orbital Plate, Laryngoscope **38**:712, 1928.

21. Moritz, S.: The Causes, Symptoms and Complications of the Diseases of the Nasal Accessory Sinuses, Brit. M. J. **1**:174, 1905.

Finally, it may be mentioned that although microscopic hemorrhages were present in practically all the internal organs, the organisms could not be seen in any but the lungs (in addition to the cranial tissues). Varney stated that "generalized infections due to *B. fusiformis* are rare," and expressed doubt concerning the cases reported by Mellon [7] and by Larson and Barron. [8] Unfortunately, the culture methods used in my case were not suitable for their cultivation, and the blood cultures were negative. Had they been positive, it might have served to confirm the belief that the pulmonary lesions were "blood-borne." In view of the presence of the organism in the diploë, from which they readily could enter the circulation, it may be inferred that a generalized septicemia with localization in the lungs accounts for their presence here. The fact that the gangrenous foci were limited to the lower lobes, however, favors instead the theory that they were aspirated via the bronchi.

Therapy.—While there are divergent opinions regarding the specificity and efficacy of arsenical preparations in the treatment of persons with infections associated with Vincent's organisms, it is fairly well established that arsenic is by far the most valuable therapeutic agent in these conditions. It was recommended by Kopetzky.[22] In Brandt's [20] case, in which the "characteristic odor" led to the detection of these organisms, recovery followed operation and the use of arsphenamine locally. He quoted others who had successfully used it both locally and intravenously. In this connection, it may be worthy of note that Varney found that in vitro *Spirilla* are more susceptible to arsenic than are the fusiform bacilli. It is suggested, therefore, that any additional efforts which may be necessary to detect the Vincent organisms in sinus infections may be worth while from the therapeutic standpoint.

SUMMARY

A case is reported in which death occurred four days after the onset of symptoms of orbital and meningeal complications due to the extension of infection from the accessory nasal sinuses. Necropsy revealed chronic infection of the frontal, ethmoid and sphenoid sinuses; osteomyelitis of the frontal bone; cellulitis of the left orbit; localized meningitis, and pulmonary gangrene. In all these locations, Vincent's organisms were present in association with other bacteria. The intracranial lesions appear to have resulted from the spreading of the infection of the sinuses through the diploë of the frontal bone, and the pulmonary lesion from aspiration of it from the throat.

22. Kopetzky, S. J.: Laboratory Aids in Otology, Laryngoscope **38**:416, 1928.

PRIMARY CHOLESTEATOMA OF THE TEMPORAL BONE *

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MILWAUKEE

In 1829, Cruveilhier [1] described a number of cases of what he named "pearly tumors." The name "cholesteatoma" was first used for these conditions by Johannes Müller in 1838 when he described eight cases. Virchow,[2] writing in 1855, regarded the condition as an epithelial new growth which he thought should be called a pearly tumor. He, together with Cruveilhier, Rokitansky and the earlier pathologic anatomists, regarded all cholesteatomas as heteroplastic new growths because these tumors, similar in all their characteristics, could be found both in the ear and in the rest of the skull attached to the pia or springing from it.

As cases of cholesteatoma of the temporal bone began to multiply, and because of their frequent association with suppuration of the middle ear, otologists began to investigate the condition more intensively. Gruber in 1862 and von Tröltsch in 1868 [2] were the first to suggest that these conditions in the temporal bone were not tumors, *sui generis,* but were processes secondary to a chronic suppuration of the middle ear. Von Tröltsch, however, could not explain how the epidermis got into the middle ear, but supposed that it was due to a metaplasia of preexisting cells in the mucosa of the attic and antral walls.

Wendt in 1873 [2] also concluded that cholesteatoma was due to a metaplasia of the existing epithelium of the mucosa of the middle ear stimulated by insults offered to it from the exterior through perforations of the drum. Wendt's theory is not largely held by otologists, because, if it were true, the greatest number of cholesteatomas would occur in cases with large central perforations, through which the mucosa of the tympanum could be most easily irritated from without. It is, however, in just this type of case that cholesteatoma rarely develops unless the perforation reaches the margo.

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* Submitted for publication, Jan. 24, 1929.

1. Cruveilhier, cited in Kuhn, A.: Das Cholesteatom des Ohres, Ztschr. f. Ohrenh. **21**:231, 1891.

2. Virchow, von Tröltsch, Wendt, Manasse, cited in Brock, W.: Die chronische Mittelohreiterung, in Denker and Kahler: Handbuch der Hals-, Nasen- und Ohrenheilkunde, Berlin, Julius Springer, 1926, vol. 7, p. 229.

In 1878, Schwartze [3] called attention to the fact that "after the healing of a chronic suppuration of the tympanum, the perforation of the drum membrane remaining open, it will frequently be seen that the epidermis of the meatus has extended into the tympanum, sometimes even into the mastoid cells." Schwartze, however, did not connect this process of repair with the cholesteatoma in question. In 1888, Habermann [4] and Bezold [5] independently came to the conclusion that cholesteatomas were almost always associated with chronic suppuration of the middle ear, and that they were the result of a migration of the epidermis of the external canal through a perforation of the drum into the middle ear, replacing there the epithelium of the mucosa of the middle ear. Habermann [4] described a case of cholesteatoma of the antrum in which the migration of the epidermis and the rete malpighii could be made out and followed both macroscopically and microscopically from a perforation and adhesion of the malleus handle and the inner tympanic wall to masses of epidermis in the antrum.

In 1880, Bezold [6] called attention to the frequent association of cholesteatoma with perforations of the membrana flaccida, and in 1891 he [7] stated that he had long been impressed with the loss of substance in the membrana flaccida in cases of tubal catarrh in the nonperforated, as well as in the perforated drums. He regarded the high grade thinning and atrophy of the membrana flaccida in these cases of tubal catarrh as offering an exceptional opportunity for a small tear or rupture due to the external air pressure, and held that adhesions between these small tears and the inner wall as the result of infectious processes might form the bridge over which the cutis could grow into the aditus region.

Bezold's statistics show that among 4,473 cases of disease of the ear in the years 1887 to 1889 there were sixty-five cases of perforations of Shrapnell's membrane, and that in forty-four of these sixty-five cases cholesteatoma could be demonstrated. He drew special attention to the triad: tubal catarrh, perforations of the membrana flaccida and cholesteatoma.

According to the Habermann-Bezold theory, the epidermis can advance into the middle ear only when the tympanic mucosa has lost its epithelium and has had this replaced by a granulating surface.

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3. Schwartze, Hermann: The Pathological Anatomy of the Ear (trans. by J. O. Green), Boston, 1878.

4. Habermann, J.: Zur Entstehung des Cholesteatoms des Mittelohrs, Arch. f. Ohrenh. **27**:42, 1888.

5. Bezold, F.: Cholesteatom, Perforation der Membrana flaccida shrapnelli und Tubenverschluss, Ztschr. f. Ohrenh. **20**:5, 1890.

6. Bezold, F.: Zur antiseptischen Behandlung der Mittelohreiterung, Arch. f. Ohrenh. **15**:1, 1880.

7. Bezold, F.: Ueber das Cholesteatom des Mittelohres, Ztschr. f. Ohrenh. **21**:25, 1891.

Manasse [7] also presupposed an inwandering of the epidermis, but differed with the Habermann-Bezold theory in that he regarded the actual proliferation of the epidermis as not due to inflammatory reaction but as an actual new growth due to the implantation of epidermoid tissue on inflamed connective tissue.

Wittmaack [8] went a step further than Bezold and saw in the deep invagination of the membrana flaccida in chronic tubal catarrh, with or without rupture, the beginning of the formation of a cholesteatoma. This invagination is into an upper portion of the middle ear closed off by a hyperplastic growth of the mucosa. This is a sort of blind sac formation. The sac continues to grow whether it is closed off or whether it has ruptured. Its epidermis then increases and builds lamellae, and this process thereafter takes on the characteristics of a cystic tumor. Wittmaack held that the formation of a cholesteatoma in the attic in this fashion can take place only when, as the result of a hyperplastic condition of the mucosa of the middle ear, the normal pneumatization has been more or less checked. Lange [9] held that the only portions of the membrana flaccida in which these invaginations can take place are the lateral marginal portions, where the epidermis is separated from the epithelium of the middle ear by only an extremely thin layer of connective tissue. Lange [9] held that even if the membrana flaccida is not invaginated in these regions, one can, as the result of inflammatory processes either external or internal, see a deep atypical growth of epidermis until it gets under the epithelium of the mucosa of the middle ear.

While the earlier authors regarded all cholesteatomas of the temporal bone as tumors due to the new growth of misplaced embryonal tissue, and otologists later adhered almost entirely to the theory that it was a secondary inflammatory result of the inwandering of epidermis, as expounded by Habermann and Bezold and amplified by their followers, the pendulum of otologic opinion is now swinging back so that the present consensus seems to be that while most of the cholesteatomas of the temporal bone are probably secondary in origin, primary congenital cholesteatoma of the temporal bone probably does occur and with about as much frequency as cholesteatomas in other portions of the skull (Hesse [10]). That these tumors do occur in other portions of the skull is apparent from the reports of Bremer,[11] Nikola-

8. Wittmaack: Ueber die normale und pathologische Pneumatization des Schläfenbein, Jena, Gustav Fischer, 1918, p. 159.

9. Lange, W.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **11**:250, 1925.

10. Hesse, W.: Zur Differentialdiagnostik wahrer und falscher Ohrcholesteatome nebst kurzer Kritik der bisher beschriebenen kongenitalen Schläfenbeincholesteatome, Arch. f. Ohrenh-, Nasen- u. Kehlkopfh. **114**:242, 1926.

11. Bremer, Georg: Ueber Cholesteatom in der Highmorshöhle, Hygiea **89**: 577, 1927; abstr. from Zentralbl. f. Hals-, Nasen- u. Ohrenh. **11**:516, 1927.

jew [12] (cholesteatoma of the antrum), Meyer [13] (cholesteatoma of the pituitary region), Otrich [14] (cholesteatoma of the ethmoid and maxillary sinuses), Marx [15] (cholesteatoma of the frontal bone) and Baily,[16] who reported on a pearly tumor growing from the cranial base into the third ventricle and on a similar tumor of the fourth ventricle.

According to Erdheim,[17] cholesteatomas of the occipital bone were described by Moller, Ogle, Morgagni, Pearson, Tannelongue and Achard and Ruschaupt. Cholesteatomas in the parietal bone were described by Franke, Blecher, Otto and Heschel; in the frontal region by Cruveilhier, Esmarch, Wotruba and Habermann, and in the inferior maxilla, by Mikulicz and Dupuytren.

Politzer [18] believed that primary cholesteatoma of the temporal bone is rare, but possible. Alexander [19] said that "primary cholesteatoma of the ear has only been observed in very rare cases as a congenital cholesteatoma of the dura which has spread to the ear by gradual increase in size." Kuhn [1] believed that cholesteatomas may arise as "new growths in the sense of Virchow." Körner,[20] writing in 1912 under the head of Tumors of the Temporal Bone, said, "True cholesteatoma of the temporal bone is, even as that of the frontal bone, the occipital bone, the inferior maxilla and the portions of the pia adjacent to the temporal bone, a heteroplastic new growth, whose origin dates to embryonal time. Genetically speaking, it has nothing to do with an ear suppuration, but is almost always complicated by such a suppura-

12. Nikolajew, N.: Ein Fall von Cholesteatom der Highmorshöhle, Russische Oto-Laryng., 1925, p. 23; abstr. from Zentralbl. f. Hals-, Nasen- u. Ohrenh. **8**: 109, 1925.

13. Meyer, Max: Ueber Geschwülste der Hypophysengegend, Würzburger Abhandl. a. d. Gesamtgeb. d. Med. **22**:127, 1925; abstr. from Zentralbl. f. Hals-, Nasen- u. Ohrenh. **8**:107, 1925.

14. Otrich, G. C.: Cholesteatomata Involving the Ethmoid Cells and the Antrum of Highmore, Illinois M. J. **48**:397, 1925.

15. Marx, H.: Ein echtes Cholesteatom des Stirnbein, Beitr. z. Anat. Physiol. Path. u. Therap. d. Ohres. **23**:273, 1926; abstr. from Zentralbl. f. Hals-, Nasen- u. Ohrenh. **9**:217, 1926.

16. Bailey, Percival: Further Observations on Pearly Tumors, Arch. Surg. **8**:524 (March) 1924.

17. Erdheim, J.: Ueber Schädelcholesteatom, Ztschr. f. Ohrenh. **49**:281, 1905.

18. Politzer, A.: Diseases of the Ear (trans. by M. J. Ballin), ed. 6, Philadelphia, Lea and Febiger, 1926, p. 417.

19. Alexander, G., in Pfaundler and Schlossmann: The Diseases of Children (English translation by Shaw and La Fetra), Philadelphia, J. B. Lippincott Company, 1914, vol. 6, p. 195.

20. Körner, O.: Lehrbuch der Ohren-, Nasen- und Kehlkopfkrankheiten, ed. 3, Wiesbaden, 1912, p. 315.

tion." And Goerke [21] said, "In spite of men like Siebenmann who have never seen such a case and doubt its existence we cannot today doubt the occurrence of true primary cholesteatoma in the tumor sense."

DIFFERENTIAL DIAGNOSIS

If one now admits that cholesteatoma of the temporal bone is not an entity but may occur there in at least two different ways, what are the distinguishing features of a primary congenital tumor?

Körner [22] established the following postulates:

1. If an epithelial tumor is found in the temporal bone and no perforation or scar of the drum exists, it is congenital.

2. It is congenital if the tumor without a preceding otitis and suppuration has produced through the labyrinth a nerve deafness years before its opening to the exterior.

3. It is congenital even if a perforation of the drum exists, if the characteristics of the latter do not make possible an inwandering of epidermis.

4. It is congenital if its fine limiting membrane has not been interrupted or broken on the side toward the tympanum.

5. If the tumor is in a location (temporal squama) into which, according to present knowledge, the epidermis never wanders, it is congenital.

6. If the suppuration of the ear has existed for only a short time and the tumor is so large that it could not possibly have grown during the period of the suppuration, it is a true congenital cholesteatoma. This is particularly true in the cases of young children.

Ruttin [23] was of the opinion that it is clinically impossible to differentiate between a primary and a secondary cholesteatoma. Ruttin held with Grünwald (cited by Hesse [10]) and Linck [24] and against Wittmaack and Hesse that the presence of abundant elastic fibers in the matrix of the cholesteatoma is a sign that it is a primary tumor. On this point, however, Hesse examined a large number of normal and pathologic temporal bones by the Weigert method, and came to the conclusion that elastic fibrous tissue is increased in all the situations where it normally occurs if the mucosa is in a hyperplastic condition, as in chronic inflam-

21. Goerke, M.: Die Geschwülste des Ohres; in Denker and Kahler; Handbuch der Hals-, Nasen- und Ohrenheilkunde, Berlin, Julius Springer, 1926, vol. 7, p. 677.

22. Körner, O.: Die eitrigen Erkrankungen des Schläfenbeins, Wiesbaden, 1899, p. 109.

23. Ruttin, Erich: Zur Pathologie des Cholesteatoms, Ztschr. f. Hals-, Nasen- u. Ohrenh. **10**:59, 1924.

24. Linck, A.: Das Cholesteatom des Schläfenbeins, in Die Ohrenheilkunde d. Gegenwart, Wiesbaden, 1914, vol. 8.

mations. Ruttin came to the conclusion that one may designate as primary: (1) cholesteatomas with perforations of Shrapnell's membrane but without otitis and (2) cholesteatomas which are closed off from the middle ear or locked in the temporal bone.

Erdheim [17] concluded from the observations in his own case as well as from those of Körner and Schwartze that primary cholesteatomas

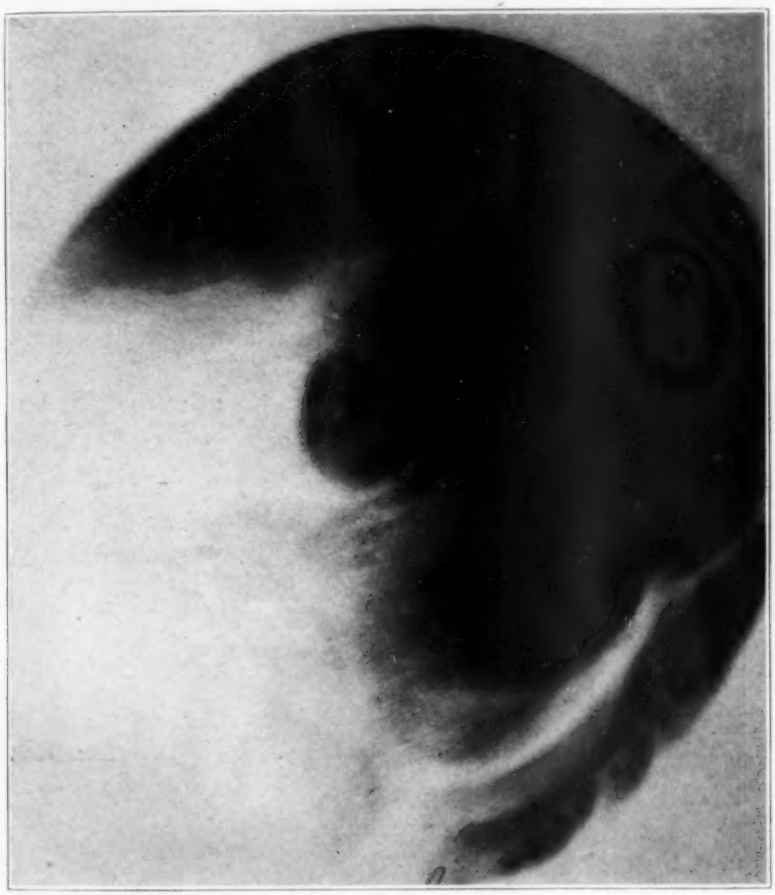

Fig. 1.—Roentgenogram of right mastoid area in a patient with primary cholesteatoma of the temporal bone. The mastoid is shown completely destroyed with obliteration of all intercellular bone septums.

have a typical location at the asterion, that is, at the region where the occipital, parietal and temporal bones join each other.

Lange [9] after cutting sections of twenty-one temporal bones with undisturbed cholesteatomas always found evidences of infection and inflammation under the epidermis, though these were at times of minor importance, and came to the conclusion that one can speak of true

cholesteatoma only when every participation of the middle ear in an inflammatory process has been excluded by a thorough clinical and histologic examination.

I thoroughly agree with the majority of otologists that by far the largest number of cholesteatomas are secondary in origin. Nevertheless, a number of well authenticated cases of primary congenital choles-

Fig. 2.—Roentgenogram of normal left mastoid in the same patient.

teatomas have been reported in the literature. They are the cases of Lucae (Linck) Kuhn,[1] Schwartze,[3] Erdheim [17] (two cases), Mondschein,[24] Manasse [24] (two cases), Berger,[24] Linck [24] (two cases), Ruttin,[23] Trible and Tibbets [25] and Rateau and Rouquette.[26] This does

25. Trible, G. B., and Tibbets, A. P.: Cholesteatoma: Case Reports, Laryngoscope **36**:512, 1926.

26. Rateau, J., and Rouquette: Le cholestéatome primitif total du rocher, Rev. de laryng. **48**:353, 1927; abstr. from Zentralbl. f. Hals-, Nasen- u. Ohrenh. **11**:146, 1927.

not in any way exhaust the literature on this subject but only the literature that has been accessible to me. To this number of cases of primary congenital cholesteatomas I should like to add the following:

REPORT OF CASE

History.—F. H., aged 18, was admitted to the Mount Sinai Hospital on April 23, 1927, from the dispensary, where she had been seen by Dr. Wolters and had been found to have a fluctuating swelling in the upper portion of the area of the right mastoid. The patient stated positively that the right ear had never discharged, so far as she or her parents knew, although the left ear had discharged at intervals for several years. The left ear had been dry for the last nine months. The swelling behind the right ear associated with some pain in this region had begun some six days before entrance into the hospital.

Physical Examination.—Dr. Wolter's dispensary note stated, "There is no discharge in the right external canal. The membrana tensa is white, rather thin

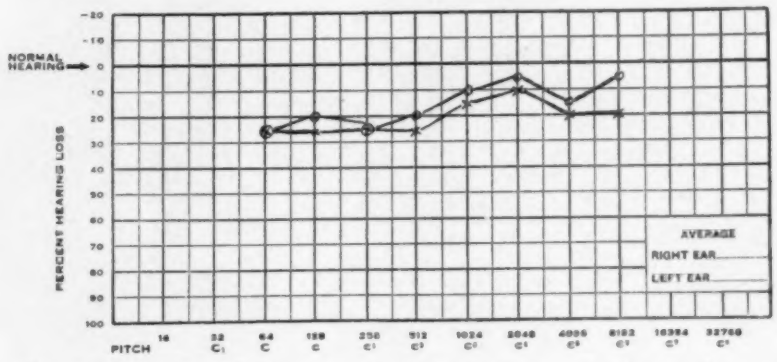

Fig. 3.—The curves obtained for the right (—X—) and left (—O—) ears in examination of the hearing with the audiometer six weeks after removal of the cholesteatoma from the right mastoid area.

and somewhat retracted. No perforations. The membrana flaccida is rather irregular but is not congested and shows no perforations." At this time, a fluctuating mass about 2 cm. in diameter was observed behind the upper attachment of the right auricle, which on being opened by the house officer, discharged a thin brown secretion. There appeared to be a perforation of the bone beneath the incision. The patient was referred to the x-ray department. The x-ray photograph (fig. 1) showed the area of the right mastoid to be completely destroyed, with an obliteration of all intercellular bone septums, while the left mastoid (fig. 2) was of normal appearance.

My examination at this time showed both tympanic membranes to be of normal appearance though slightly retracted. Perforations were not seen in either drum. All the landmarks of the drum were of normal appearance. There was no congestion or swelling of either drum. The hearing was roughly tested in the patient's room; a quiet pocket watch was heard at 8 inches (20.3 cm.) with each ear, while a whisper was heard at more than 15 feet (38.1 cm.) with each ear. The Pirquet and Wassermann reactions were negative. Examination of the blood showed: red cells, 4,200,000; white cells, 9,400, and hemoglobin, 75 per cent. The

differential count showed: polymorphonuclears, 62 per cent; esophinophils, 1 per cent; small leukocytes, 30 per cent; large leukocytes, 4 per cent, and transitional, 3 per cent. The urine showed a trace of albumin, but was otherwise normal.

Operation, April 25, 1927.—There was a draining fistula about 2 cm. back of the upper attachment of the right auricle where the previous incision had been made. The usual postauricular incision was made to the bone. In separating the

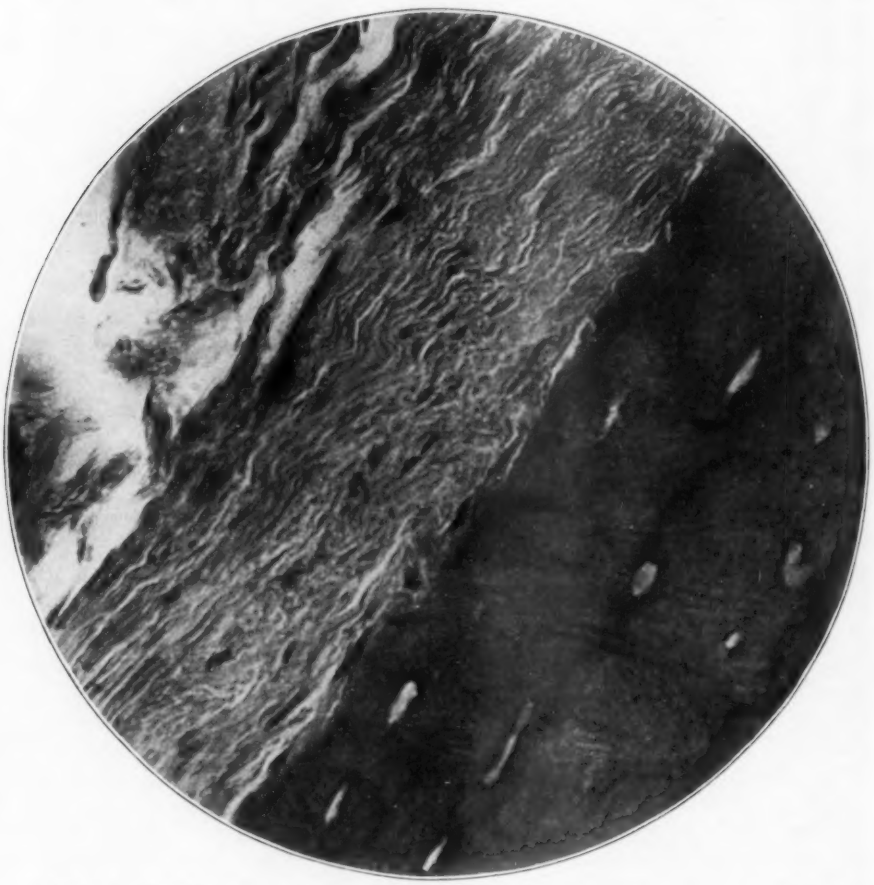

Fig. 4.—Section of the soft material of the cholesteatoma as seen under the microscope.

fibers of the sternocleidomastoid muscle from the tip with a sharp elevator, the tip was broken into. The cortex was extremely thin and soft over the entire area of the mastoid. When this was removed, the entire mastoid cavity was found filled with cholesteatomatous masses. This process had laid bare the lateral sinus from above the knee to the jugular bulb and had so flattened it that its outlines could be made out only with difficulty. The bone over the cerebellar dura behind the sinus had been eroded over an area as large as a half dollar. A small cupola-like extension of this general cavity toward the region of the mastoid antrum was cleaned out. This, however, was found to be absolutely shut off from the middle

ear cavity by a partition of solid bone, which would nowhere admit a probe. All the walls of this enormous cavity were as smooth as burnished metal. The wound was packed as in a simple mastoid operation, and the middle ear was not entered.

The postoperative course was entirely uneventful, and the patient was dismissed from the hospital on May 5, 1927. The mastoid wound was completely healed in about six weeks. At that time, an examination of the hearing with the

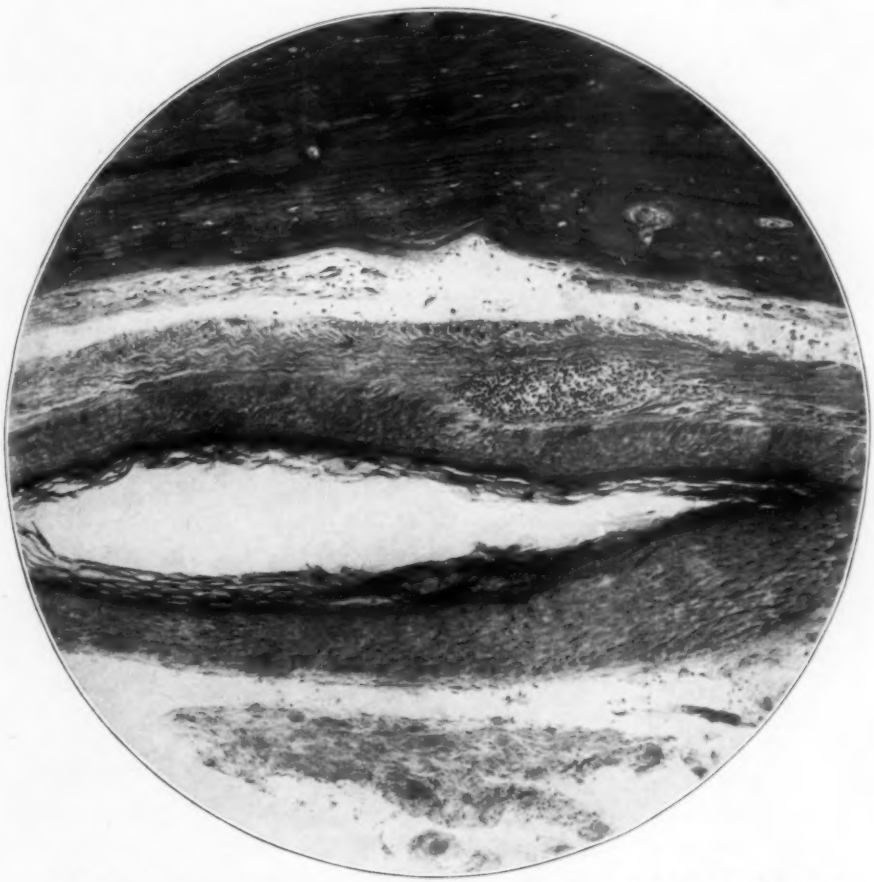

Fig. 5.—Section of the bone fragment in cholesteatoma of the mastoid, as seen under the microscope.

audiometer revealed a symmetrical curve for both ears with some reduction of hearing in the lower tone range (fig. 3).

Histologic Examination.—Most of the tissue was soft, gray and structureless, and was arranged in concentric layers. The remainder of the tissue was made up of fragments of bone. A direct smear of the tissue failed to reveal crystals of cholesterol. Sections of the soft material showed microscopically only a structureless, rather crystalline material. Sections of the bone fragments showed an increase in density of the bone with the bone cells small or widely separated. The periosteum was thickened. The surface was covered by a thin layer of epithelium,

squamous in type, and this was covered by a thin layer of the same material as that removed from the cavity (figs. 4 and 5).

Diagnosis.—The condition was diagnosed as cholesteatoma of the mastoid.

CONCLUSION

This case seems to be one of primary congenital cholesteatoma of the temporal bone. It can never be classed as absolutely such because a histologic examination of the drum and the middle ear was not possible. It seemed to have developed in the region of the asterion. No history of any preceding disease of the right ear was obtainable, either from the patient or from her parents, although the patient reported attacks of otitis of the left ear. The right drum was intact and did not show any signs of perforation or scar. The hearing was good, although it showed some loss in the lower range. The middle ear was entirely shut off from the cavity of the cholesteatoma by a firm bony wall in the aditus region. For these reasons, in spite of the lack of histologic study of the middle ear, I must assume that this was a case of true cholesteatoma. The process was insidious, symptomless and destructive, as Lillie and Stark [27] have pointed out, and was brought to the attention of the patient only by the swelling on the side of the head, which she did not connect with the ear. As to the method of operating on this, as in uncomplicated mastoiditis, without opening into the middle ear cavity, I believe it was justified by the normal appearance of the drum and the middle ear, by the absolute absence of any connection between the middle ear and the cavity being operated on and by the result. Karlefors [28] also reported a case of cholesteatoma with an intact drum, in which he did a modified radical operation without any plastic work and in which healing and good hearing resulted in about four weeks.

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27. Lillie, H., and Stark, W.: The Insidious, Symptomless and Destructionless Effect of Cholesteatoma, Surg. Clin. N. America **6**:1359, 1926.

28. Karlefors, J.: Cholesteatom mit unverlelzterm Trommelfel. Radikal Operation ohne Plastike Heilung, Hygiea **89**:855, 1927; abstr. from Zentralbl. f. Hals-, Nasen- u. Ohrenh. **12**:105, 1928.

ESOPHAGOSCOPIC STUDY OF ESOPHAGEAL VARICES

REPORT OF TWO CASES OF ESOPHAGEAL VARICES PERSISTING AFTER SPLENECTOMY *

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Before the introduction of a direct method of examining the esophagus, diseases of that organ were diagnosed by inference; and lesions that failed to produce physical signs, altered function or characteristic roentgen appearances, from which the existence of the lesion might be deduced, remained unrecognized. Such was the case with esophageal varices. But with the advent of the esophagoscope, the esophagus was no longer "terra incognita." Every portion of the interior of the organ became accessible to direct examination. This advantage has enabled the discovery and definite diagnosis of many pathologic conditions which, prior to the general use of the esophagoscope, went unsuspected. It permits of a degree of accuracy in the diagnosis of disease that is attained only when the diseased area can be directly inspected. With preliminary roentgenologic study, a diagnosis can be made by the esophagoscope more readily, with greater certainty and with more safety to the patient than by any other means of examination. The necessity of employing esophagoscopy for the adequate study of lesions of the esophagus has frequently been demonstrated,[1] but it is particularly pertinent to emphasize the value of endoscopy in the consideration of esophageal varices, for it is only through direct examination that they can be definitely recognized,[2] and in their presence blind methods of examination are especially dangerous.

Varices of the esophagus are not regarded as a frequent lesion. Certainly they are a lesion not frequently seen by endoscopists. This must be due, in part, to the fact that they may remain symptomless for years. It is only when they are the source of considerable hemorrhage that they attract the attention of the internist and suggest

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1. Jackson, Chevalier: Carcinoma and Sarcoma of the Esophagus: A Plea for Early Diagnosis, Am. J. M. Sc. **169**:625 (May) 1925.

2. Gottenstein, G., in Keen: Surgery, Philadelphia, W. B. Saunders Company, 1918, vol. 3, p. 793.

to him the advisability of an esophagoscopy for diagnosis. But in actual occurrence, varices must not be infrequent. Necropsy reports are the only data obtainable in this regard, for an insufficient number of cases of varices of the esophagus have been studied endoscopically during life to justify conclusions as to their relative incidence thereby. Necropsy observations show some variation, which can be explained on the basis of difference in technic of performing the examination and by the fact that improved methods of treating the underlying conditions responsible for varices bring patients so affected to autopsy later now than heretofore. Preble,[3] in 1900, reporting sixty cases of fatal gastro-intestinal hemorrhage, found that esophageal varices were present in 80 per cent, and in one half of these macroscopic ruptures of the varicose veins were found. He added that many more varices of the esophagus would be discovered at necropsy if fluids were injected to detect them. In 1920, Blumenau[4] reported that of 126 patients affected with obvious portal cirrhosis at the time of death, 19 per cent died of vascular lesions, of which esophageal varices were a very important part. McIndoe[5] recently reported that of twenty-six patients who died of advanced portal cirrhosis, 50 per cent died of gastro-intestinal hemorrhage, and in all cases in which gastro-intestinal hemorrhage occurred, careful examination invariably revealed well developed esophageal varices. He expressed the belief that recently improved methods of treatment in cirrhosis of the liver, the most frequent underlying cause of varices of the esophagus, prolong the life of cirrhotic patients and permit the vascular complications of cirrhosis to progress further than formerly occurred. He stated that lesions of the vascular system are now responsible for about 50 per cent of the deaths in cases of portal cirrhosis, and of such lesions by far the most frequent and serious are esophageal varices with ulceration. Older observers, Dusaussay,[6] Chautemps[7] and Gilbert and Villaret,[8] also emphasized the importance of esophageal varices in gastro-intestinal hemorrhage and pointed out the serious prognostic significance of this complication. McIndoe, Preble and others stated the belief that at

3. Preble, R. B.: Conclusions Based on Sixty Cases of Fatal Gastro-Intestinal Hemorrhage Due to Cirrhosis of the Liver, Am. J. M. Sc. **99**:263, 1900.

4. Blumenau, Ernst: Ueber Todesursache bei Lebercirrhose, Arch. f. Verdauungskr. **27**:1, 1921.

5. McIndoe, A. H.: Vascular Lesions of Portal Cirrhosis, Arch. Path. **5**:23 (Jan.) 1928.

6. Dusaussay, M.: Étude sur les varices de l'oesophage dans la cirrhose hépatique, Thèse de Paris, 1877.

7. Chautemps, E.: Des hémorrhagies dans la cirrhose du foie, Thèse de Paris, 1875.

8. Gilbert, A., and Villaret, Maurice: Les circulations veineuses supplémentaires de la paroi thoraco-abdominale antérieure, Rev. de méd. **27**:305, 1907.

necropsy, varices of the esophagus are apt to be overlooked unless special care is taken to detect them by inflation or by injection. Even in the most marked cases the veins collapse after death and are practically invisible beneath the squamous epithelium of the esophagus. The points of rupture are usually inconspicuous, and for this reason, also, the varices are often overlooked in the routine autopsy. But if inflation or injection is resorted to, the varices stand out with startling prominence. It seems probable that the occurrence of varices of the esophagus is more common than is generally believed.

In life, aside from a deductive diagnosis, which is largely a matter of surmise and conjecture, they remain undiagnosed until esophagoscopy is done. The two cases herein reported did not present any symptoms, physical signs, roentgen or other evidence from which a diagnosis of varices of the esophagus could be made, except by esophagoscopy. The recognition of varices, even though they never produce any sort of symptom, is of more than theoretical importance, as fatal results have followed an initial sudden hemorrhage from previously unrecognized esophageal varices.[9]

The lower portion of the esophagus contains an abundant network of veins, draining chiefly into the coronary gastric vein, which empties into the portal vein directly or into the splenic vein near its junction with the portal.[10] Veins of the lower esophagus drain to a lesser extent into the azygos and hemiazygos veins, and form small anastomoses with the diaphragmatic and intercostal veins. Actual or potential union between the portal and caval venous systems is therefore formed through the esophageal veins.

The various pathologic conditions of the upper part of the abdomen, notably of the liver and spleen, which may indirectly produce esophageal varices are too well known to require description, but portal cirrhosis with its resultant portal obstruction, cancer of the liver, chronic passive congestion, pressure on the portal vein by tumors, inflammatory exudates, enlarged glands, etc., may be mentioned. Varices of the esophagus may coexist with the common dilatation of the venous system in which the veins of the legs, scrotum and rectum are most conspicuously affected.[11] Enlarged, tortuous veins are also not uncommonly associated with the sacciform dilatation of the esophagus above the hiatus, in cases of so-called cardiospasm.[12] Splenomegaly, with

9. Playfair, Ernest: Lancet **2**:1079 (Dec. 13) 1919.

10. Spalteholz, W.: Atlas of Human Anatomy, Philadelphia, J. B. Lippincott Company, 1922.

11. Jackson, Chevalier: Bronchoscopy and Esopagoscopy, ed. 2, Philadelphia, W. B. Saunders Company, 1922, p. 369.

12. Thomson, St. Clair: Diseases of the Nose and Throat, ed. 3, New York, D. Appleton & Company, 1918, p. 647.

increased flow of blood from the spleen and resultant portal congestion, produces a related engorgement of the portal venous tributaries, and the lower esophageal veins are apt to be the ones most extensively involved. With a long continued state of dilatation, the esophageal veins permanently increase in size and length and assume marked tortuosities. The factors favoring the development of esophageal varices in the presence of portal obstruction need not be fully discussed. The proximity to the seat of obstruction and the comparative directness of the communication of the portal with the caval systems through the esophageal veins no doubt favor this over others as an anastomotic pathway. The lack of support offered by the submucosal tissue to veins in this situation together with the aspirating effect of the negative intrathoracic pressure with respiration [5] and the incompetence of the valves in adult life are no doubt responsible factors. In any event, of all the collateral channels developed as a result of portal venous hypertension, the lower esophageal veins are most important because of the frequent association of serious gastro-intestinal hemorrhage with them.

Varices of the esophagus, lacking an inflammatory basis, produce little pain or discomfort, and affect the functional efficiency of the esophagus slightly or not at all. Hemorrhage is the most common and important symptom. The varices may remain unsuspected over a period of years until a frank hemorrhage with hematemesis or passage of tarry stools directs the attention of the patient to his condition,[13] or repeated slight hemorrhages with passage of the blood into the stomach and subsequent vomiting of "coffee ground" flocculi simulate a typical feature of cancer of the stomach and tend to mislead the diagnostician. A notable example of this error has been reported by Jackson.[14] A man who presented many of the typical features of cancer of the stomach, in respect to history, age, cachexia, "coffee ground" vomitus, etc., was subjected to a laparotomy for the purpose of exploring the abdomen, with negative results. If a single direct esophageal examination had been made prior to the operation, the laparotomy could no doubt have been avoided. For at the first endoscopy by Dr. Jackson, a diagnosis of bleeding esophageal varices was made. Under local and general medical treatment the patient became symptom free within a few weeks, recovered his health completely, and the diagnosis of varices of the esophagus as the cause of hematemesis was proved to be correct.

A routine physical examination is of value in the diagnosis of varix in that it often reveals the pathologic condition which underlies

13. McCrae, T., in Osler: Principles and Practice of Medicine, ed. 10, New York, D. Appleton & Company.

14. Jackson, Chevalier; Tucker, Gabriel; Clerf, L. H.; Lukens, R. M., and Moore, W. T.: Hematemesis: Plea for Objective Methods of Diagnosis, J. A. M. A. **85**:870 (Sept. 19) 1925.

the congestion and dilatation of the esophageal veins. An enlarged liver in an alcoholic subject, ascites, an enlarged spleen, varicosities of the hemorrhoidal or of the superficial veins of the abdominal wall and a caput medusae are physical manifestations which coupled with bleeding from the upper gastro-intestinal tract may lead to a deductive diagnosis of varices of the esophageal, gastric or duodenal tributaries of the portal vein. But this deductive method of diagnosis is inconclusive and uncertain. At best, it can only localize the varices somewhere in the upper tributaries of the portal system, whereas by esophagogastroscopy, reliable information regarding the esophagus and cardiac end of the stomach can be had. An esophagoscopic examination can be done with safety and with little loss of time, frequently without hospitalization. It is therefore reasonable to urge, when from clinical study portal cirrhosis or other lesion is found from which resultant portal obstruction may be suspected or anticipated, that an esophagoscopy be done to determine whether back pressure in the esophageal veins exists. Then steps can be taken to combat the varices, if present, before they have reached a great size and before they have ulcerated and produced symptoms. It is better to be aware of the presence of varices before hemorrhage than after. The esophagoscopy can be done satisfactorily without the use of any anesthetic, general or local, and with little discomfort to the patient.

ENDOSCOPIC APPEARANCES

Varices are encountered most frequently about the cardiac orifice of the stomach and in the lower third of the esophagus, down to which level the usual pale, smooth folds of mucosa are seen. The beginning of the area involved in the varicose formation is usually well defined. Well developed varices appear as irregular tortuous and rounded nodular elevations on the surface of the folds, covered by mucosa and possessing a fairly marked dark blue color. On pressure, they are rather easily compressible. They present a striking resemblance to clumps of small hemorrhoids. Varices which appear to be in a less advanced stage are seen also as dilated, bluish vessels, having a sinuous course, lying close under the mucosa of the esophagus through which they stand out vividly. These veins lack the nodular, bulbous character, but are uniformly and greatly dilated. They resemble sclerotic temporal vessels in their tortuosity. Erosions may occur on the surface of the varices, determining the presence or absence of hemorrhage, which may be petechial or free and profuse. It is surprising what a great amount of hemorrhage may occur from erosions of small size and of apparently little importance.

TREATMENT

Prophylactic.—The earliest symptoms of esophageal varices occur late in the course of the lesion. It is only after ulceration has taken place that varicose veins, through conspicuous hemorrhage into the gastro-intestinal tract, call attention to their presence. Whatever treatment for the varices is given then is given late. To be early, treatment must be instituted before symptoms have occurred.

It is true that in some cases there may be no indication of the existence of varices so long as they remain symptomless. In some cases, the first evidence of the underlying pathologic change producing the varix is hemorrhage into the gastro-intestinal tract. But in many cases the underlying pathologic condition can be diagnosed on the basis of other clinical observations and the existence of varices suspected or anticipated long before they produce symptoms. If in every case in which portal obstruction is suspected on clinical examination the benefit of an early esophagoscopic examination were given, many cases of varices would no doubt be discovered while still symptomless, and treatment could be instituted. Many such patients, treated with the same measures which prove unsatisfactory when ulceration has already occurred, would likely be spared the serious complications of ulceration and hemorrhage.

As noted at endoscopic examination and also at necropsy, the varices project prominently from the mucosa of the esophagus, and are susceptible to trauma. A soft, nonirritating diet, avoiding extremes of heat and cold in food and liquids should be ordered, and all solid food eaten slowly so that boluses may be small and well masticated. The use of bougies, stomach tubes and other blind methods of examination is particularly contraindicated. The patient should be allowed a minimum of exertion in order not to raise the blood pressure. The underlying pathologic condition responsible for the development of the varices should be treated by whatever medical or surgical measures are most suitable. The decision in this regard depends on the site, size and condition of the varices. Since this can be determined only by endoscopy, a direct esophageal examination must be made preliminary to any definite plan of treatment.

Treatment for Bleeding Varices.—After hemorrhage has occurred, even though slight, the patient should be put at complete rest for a time to permit healing of the ulceration. The diet should be liquid, and the stools or vomitus examined for blood. Bismuth subnitrate, given dry on the tongue and dissolved slowly, should be administered at frequent intervals for the local hemostatic and healing effect on the ulcerations present in the varices. Endoscopic examinations may be done as indicated to determine the progress of the case. The general

treatment is governed by the nature of the underlying pathologic condition producing the varices.

Surgical Treatment.—It is not within the scope of this paper to discuss the sugical treatment for varices of the esophagus, but since esophagoscopy is the means of early diagnosis of varices, it seems relevant to mention that surgical treatment is much more effective if given early than late. Probably the most popular method of dealing with portal hypertension has been the Talma-Morison operation with its modifications, such as the production of adhesions between the various abdominal organs and the abdominal wall. All of them rely for success on the slow development of new anastomotic vessels in the adhesions so formed. The time required may be much too long for the patient to derive any benefit from them,[15] unless they are done early.

Another surgical measure is that of splenectomy for the purpose of reducing the amount of blood entering the portal circulation, for the spleen contributes about 20 per cent of the total amount of portal blood. Once the esophageal veins have become permanently varicose and ulcerated, the chances of obtaining the result desired, as indicated by the course of the two cases described herein, are much less apparently, than if the same procedure had been done earlier.

REPORT OF CASES

The following cases are of interest in that in each case marked esophageal varices were found in patients on whom a splenectomy had been performed several years previously, and in whom bleeding from the gastro-intestinal tract continued unchecked after the splenectomy.

CASE 1.—I. L., a man, aged 28, presented a history of bleeding from the gastro-intestinal tract. The bleeding first occurred ten years before, when at the age of 18, he was employed in the navy. He had been in good health up to that time, and there was nothing of note in the medical history except that in childhood he had had frequent epistaxis. The family history and social history were unimportant, and contained no suggestion of hemorrhagic diathesis. The first hemorrhage from the gastro-intestinal tract occurred in October, 1918. At that time, while doing heavy work in the Navy Yard, he noticed a dull pain in the left hypochondrium which did not force him to stop work and did not cause much concern. One or two days later, he noticed dark, tarry stools. After several days in bed there were no further symptoms, and he resumed work until a short time later when he suddenly became nauseated and vomited about a quart (946 cc.) of dark blood. At this time he was taken to a hospital, and the diagnosis of peptic ulcer was made.

In 1920, he was admitted to the University Hospital, where a diagnosis of splenic anemia and chronic appendicitis was made. Examination of the blood indicated a fairly marked secondary anemia. A splenectomy and appendectomy

15. Ducamp, Carrier et al.: Hémorrhages dues à Varice Oesophage, Bull. Soc. d. sc. méd. et biol. de Montpellier **3**:139, 1921.

were done in July, 1920. At the time of this operation, varicosities about the stomach and duodenum were not noted; the liver appeared normal. The condition of the spleen favored the diagnosis of Banti's disease.

After the splenectomy, the patient had about a dozen hemorrhages, on an average of every eight months, and varying in amount from 1 or 2 ounces (30 or 59 cc.) to 1 quart. Before bleeding occurred, he frequently had a stabbing frontal headache and a feeling of nasal obstruction. At times he noticed tarry stools for a day previous to an attack of hematemesis. After bleeding, he would feel well again. Occasionally, transient attacks of jaundice followed. Excitement and exertion appeared to be exciting causes of the hemorrhage.

After the splenectomy, the patient was admitted to the hospital seven times because of hematemesis or passage of tarry stools. The hemoglobin fell as low as 41 per cent, and he received two transfusions. Each time he was admitted to the hospital studies of the blood were made, which indicated the presence of a

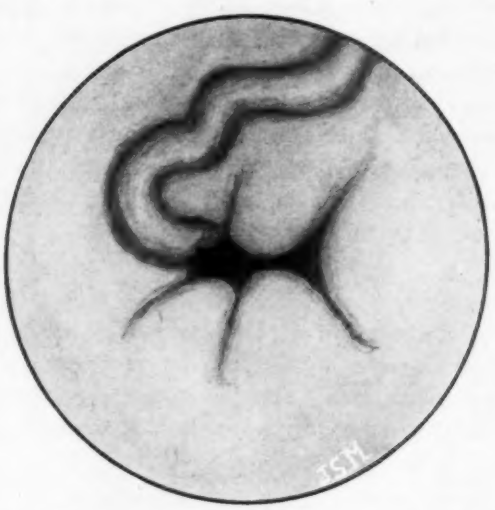


Fig. 1 (case 1).—Endoscopic view, showing a large, dilated, tortuous vein in the lower esophagus, running in the axis of the lumen.

secondary anemia. The coagulation and bleeding intervals were not lengthened. Some examinations indicated an increased fragility of the red cells. Serologic tests were negative.

Each time, the patient improved under a regimen of rest and symptomatic treatment. The hemorrhages ceased, and he usually left the hospital within two or three weeks. After each discharge, he would go home and remain in apparent good health for about six months, and then bleeding would occur again. He never had fever or prolonged jaundice. He had no symptoms referable to the gastro-intestinal tract aside from the bleeding, no hemorrhoids and no history of purpura. There was no loss of weight; his appetite was good and in general he felt quite well. His latest hemorrhage was in January, 1928, one and one-half months prior to the esophagoscopic study.

Physical examination in February, 1928, revealed little of note. He was well developed and nourished and only slightly pallid. The chest and abdomen were apparently normal; the temperature, pulse and respiration were normal. Blood

pressure was 110 systolic; 64, diastolic. A slight thyroid enlargement appeared to be a simple colloid goiter. Examinations of the blood indicated a mild secondary anemia. The bleeding and coagulation tests gave results within normal limits. The Wassermann reaction of the blood was negative; the urine was normal. The roentgenologic examination of the gastro-intestinal tract gave negative results, except for an irregularity of the duodenal shadow, probably due to adhesions.

A diagnostic esophagoscopy done in March, 1928, quickly revealed the presence of esophageal varices. The mucosa of the hypopharynx and upper esophagus was normal. At a point 29 cm. from the upper incisors, on the lateral and anterior walls of the esophagus, was found a distended, congested vessel, a varicose vein, lying close under the mucosa. It had a definite deep blue color, and followed a tortuous course down the axis of the esophagus. Proceeding farther down the esophagus, at irregular intervals, for a distance of about 8 cm., similar vessels were encountered. They presented marked tortuosities, and some had a distinct

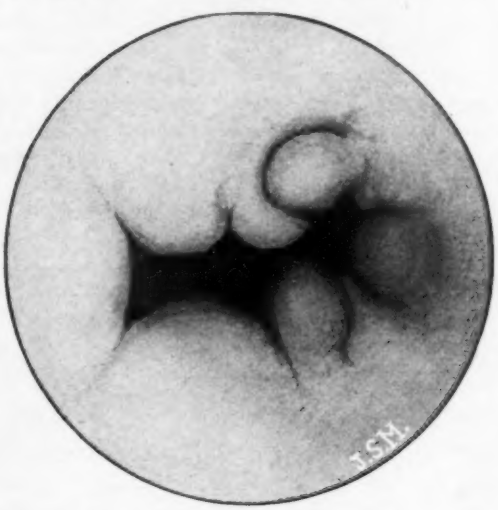


Fig. 2 (case 2).—Endoscopic view, showing marked venous tortuosities in the lower thoracic and abdominal esophagus.

bluish appearance. The abdominal esophagus and cardiac orifice of the stomach exhibited similar varices. No definite ulceration or bleeding point was found, but appearances suggested previous hemorrhage from this site.

Case 2.—S. W., a man, aged 24, presented a history of hematemesis dating back to 1917. His family history and social history were unimportant, containing no suggestion of a hemorrhagic diathesis. At the age of 7 years he had scarlet fever, following which he was never strong. In 1914, enlargement of the spleen was discovered. About two or three years later, he had his first gastro-intestinal hemorrhage. Following this, the patient continued to have hemorrhages with occasional vomiting of blood and passage of tarry stools at irregular intervals. In 1918, a diagnosis of splenic anemia was made and a splenectomy performed. At the time of operation, some engorged veins were found about the stomach and duodenum. The pathologic examination of the spleen corroborated the clinical diagnosis. The gastro-intestinal hemorrhages continued unchecked after the splenectomy. In 1923, the patient returned to the University Hospital for further

study. At that time examinations of the blood showed a secondary anemia and a normal bleeding and coagulation time; there was a normal output of urobilin in the feces. In 1926, at another clinic, a diagnosis of duodenal ulcer was made, and the abdomen opened. No ulcer was found, but varices around the pyloric region of the stomach were reported. Hemorrhages have occurred irregularly since then. On one or two occasions, they have been precipitated by injury: once by a blow in the face and once after the patient was shaken up in an automobile accident. The latest hemorrhage was in January, 1928, one and a half months prior to the esophagoscopy.

Physical examination in February, 1928, indicated a secondary anemia; otherwise, there was little of note. The temperature was normal, the respirations and pulse slightly increased. The serologic tests were negative. On roentgen examination, there was slight obstruction to the capsule found at the lower end of the esophagus; the pylorus and duodenum showed the presence of adhesions; otherwise, the examination was negative.

An esophagoscopy was done in March, 1928, and the presence of marked venous varicosities demonstrated in the lower thoracic and abdominal esophagus. The mucosa of the esophagus was pale and somewhat thickened. At a point in the lower esophagus about 32 cm. from the upper incisors, varices were first encountered. They appeared as nodular enlargements, irregular and tortuous in character, lying close beneath the mucosa of the esophagus. They varied in size from that of a pea to that of the end of the little finger. No pulsation was seen in the varices. A distinct bluish color was noted in several of them. The similarity of the appearance of the varices to that of hemorrhoids was striking. Farther down in the thoracic and abdominal esophagus, varices were also observed. There was no positive evidence of hemorrhage or ulceration, but appearances suggested the possibility of previous hemorrhage from this site.

COMMENT

Varices of the esophagus must always be considered as a possible source of hemorrhage into the gastro-intestinal tract. They are the most frequent cause of hematemesis in splenic anemia. A direct esophageal examination is necessary to demonstrate their presence. The size of the veins, the extent of the area involved and the existence of ulcerations and hemorrhage are important features to be determined. In the cases here reported, the latest hemorrhage occurred about one and one-half months prior to the esophagoscopy. At the time of the esophageal examination there was no positive proof that bleeding had occurred from the varices of the esophagus, but it is to be recalled that the site of considerable hemorrhage may be small and inconspicuous. The local appearances suggested that the esophageal veins had likely been the site of previous bleeding.

In these cases, the esophageal varices appear to have developed secondary to the splenomegaly. The probable reason for their presence after the splenectomy cannot be determined by endoscopy, and the question, therefore, will not be discussed at length. It may have been due to portal obstruction resulting from cirrhosis of the liver, which is commonly associated with splenic anemia; or, permanent dilatation

of the esophageal veins having occurred and the blood flow thoroughly established to the caval system, the veins may have continued to function in the reverse to normal direction, in spite of the removal of the spleen.

The results of esophagoscopy are important to the surgeon in cases similar to these. He is confronted with the question of laparotomy for the eradication of the bleeding varicose veins, or for the production of new adhesions in the abdomen, and additional collateral circulation thereby. If endoscopy demonstrates the absence of esophageal varices, then it is more probable that the veins about the stomach and duodenum are the source of the hemorrhage. On the other hand, if well developed esophageal varices are found and are the site of hemorrhage, the hope of eradicating the condition by laparotomy is less bright.

Clinical Notes

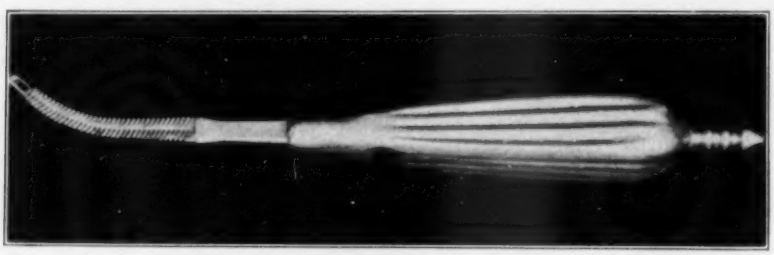
AN IRRIGATING CANNULA-RASP FOR MAXILLARY SINUSITIS *

D. M. Butler, M.D., Boston

This new instrument is used to take the place of several instruments usually employed in the diagnosis and treatment for acute and chronic conditions of the maxillary sinus.

The treatment for infections in this sinus depends on establishing a sufficient opening in the naso-antral wall to allow proper drainage, with or without irrigation.

The instrument described is made for the purpose of supplying an irrigating cannula for puncturing and washing out the antrum com-



Irrigating cannula-rasp for maxillary sinusitis.

bined with a rasp that will remove as much of the naso-antral wall as the operator deems necessary. In this way the common operations of puncturing and then removing a portion of this wall are simplified.

The instrument is made of tempered steel; it tapers to a fairly sharp point as on an ordinary trocar, having an opening on two sides for irrigation. It is slightly curved and extends to a widened portion or stop, separating the cannula from the rasp. The rasp is graduated and square and has rasping teeth on all sides. It has a good-sized handle with a corrugated nozzle for a rubber tube attachment.

TECHNIC

The nose is cocainized by placing a pledget of cotton soaked in 5 or 10 per cent cocaine in the inferior meatus, and another over the inferior turbinate, which are allowed to remain for fifteen minutes. The instrument is placed in the nose, so that the trocar point comes under the inferior turbinate. When this is not

* Submitted for publication, Aug. 13, 1929.

possible, fracture of the turbinate upward is advised. With firm pressure, upward and outward, the naso-antral wall is punctured; the cannula portion up to the stop enters the antrum. The sinus can then be irrigated.

If the condition found in the sinus necessitates any further treatment, without removing the instrument and with slight additional pressure, the rasping portion is forced into the antrum. With a backward push, then a forward sawing motion, a large opening is made in the wall. As repeated puncturing is unnecessary, irrigations can be continued with the use of an ordinary silver eustachian catheter with rubber tube attached.

COMMENT

This procedure is not advocated in any way to replace the radical antrum operation, but I have found that such operative intervention was necessary only in cases of new growths and polyposis of the antrum.

The advantages of the technic with the irrigating cannula-rasp are:

1. The ease of performance, with local anesthesia used in adults and gas in children.

2. Irrigation and opening in the naso-antral wall made with same instrument at one sitting.

3. The opening in the naso-antral wall made large enough for sufficient drainage and continued irrigations.

This instrument is made and sold by Codman & Shurtleff Company, Boston.

A NEW OPERATING ATTACHMENT FOR CATHETERIZING THE EUSTACHIAN TUBE*

ROBERT M. COLBERT, M.D., NEW YORK

The introduction of catheters, bougies and electrodes into the eustachian tubes has become more and more a vital factor in the treatment of diseases of the middle ear. Dilatation of the collapsed tube, diathermy in chronic catarrhal deafness, fulguration of polypi at the orifice and other such allied conditions require accuracy not only of application to the local area, but also a maintenance of this application during the entire period of treatment.

The instrument used for many years to visualize the posterior nasal cavities is the nasopharyngoscope, which is merely a modification of the original Nitze cystoscope. In fact, there is hardly a visualizing instrument in nose and throat practice which has not been first developed by the urologists.

In 1921, I first became interested in developing a cannula to maneuver for direct application to the eustachian orifice, and it was not until 1923, through the aid of Mr. Reinhold H. Wappler, that I developed a cannula which could be attached to the nasopharyngoscope by means of a collar and could be used to guide a catheter under vision. This cannula had an equal curve at each end so that one end could be used for the right side and the other for the left side.

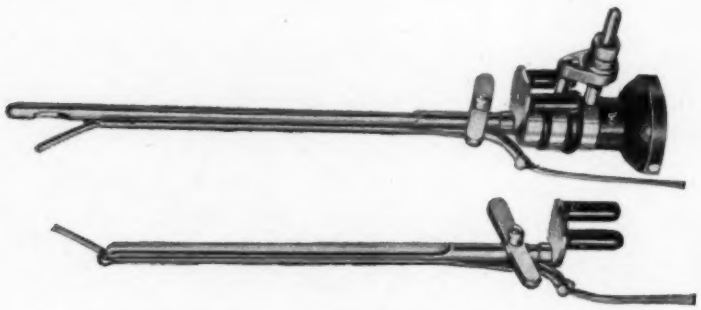
* Submitted for publication, June 4, 1929.

However, due to the flimsy manner of attachment and to the fact that, once inserted, there was no definite external guide to the various positions of the catheter, lens, degree of catheter angle, etc., in relation to the eustachian orifice, I did not feel that the instrument was sufficiently developed for general use.

Now, however, through the assistance of Mr. Wappler, I have perfected an instrument which, I believe, is so accurately arranged that in catheterizing the eustachian tube it can easily be successfully employed by any one having little or no experience.

The instrument consists of a tube through which the nasopharyngoscope may pass, and to which the latter is locked in position by a pair of forked prongs. This makes the combination of the attachment and its scope one fixed unit. Besides this, there is another tube through which the catheter, electrode, etc., may pass. At the end of this tube, there is an obturator which will deflect the catheter at the point of emergence, not only either to the right or left at will, but in various degrees of deflection on either side.

This obturator is controlled by a little lever at the external end of the instrument. This lever is always in sight and is pushed forward toward the lens on the same side toward which it is intended for the catheter to emerge. Then, by



New operating attachment for catheterizing the eustachian tube.

moving this lever, one can regulate the amount of deflection desired on that side at will and under constant vision.

Likewise, the illuminating telescope has a marker on the eyepiece in the form of a little knob, which indicates the position of the lens. When the knob is placed at 4 o'clock (the eyepiece being used as the dial of a clock), the lens and illuminating lamp are in position to view the left eustachian orifice, while if the knob is placed at 8 o'clock, the position is set for procedure at the right orifice. The knob may then be moved at will to enlarge the field of vision about the orifice.

As a result, even though the instrument is inserted into the nose, there is an accurate guide on the outside to indicate the various positions of the catheter and lens which are hidden from view.

Another important feature is the fact that this attachment has no curve at the end which is introduced into the nose, thereby maintaining as small a diameter as possible and allowing easy introduction into an extremely narrow nasal passage.

A simple working technic is thus developed wherein the eustachian orifice can be quickly and safely probed, catheterized, fulgurated, treated with diathermy or otherwise treated under the guidance of constant vision.

160 West Eighty-Seventh Street.

OBSERVATIONS ON THE OCCURRENCE OF LUNG ABSCESS FOLLOWING OPERATIONS ON THE TONSILS: REPORT OF TWO CASES*

MARGARET NOYES KLEINERT, M.D., BOSTON

In the two cases of abscess of the lung under consideration, the patients had entered the New England Hospital for Women and Children previously for tonsillectomy which was performed on the laryngologic service.

The first case occurred at about the time Richardson[1] wrote his early observations on abscess of the lung in 1912. Since that time the number of cases reported has reached more than 400. In 1922, Moore[2] collected 202 unreported cases, obtaining his information from a questionnaire sent to 1,020 specialists in diseases of the nose and throat throughout the country.

For some time it was thought that abscess of the lung occurred only in patients operated on under general anesthesia, but a certain percentage of cases has been found to have occurred under local anesthesia; this was shown in the report of Ochsner[3] in the use of his "passive" technic. In fact, any anesthesia that completely abolishes the reflexes of the throat seems to be a large contributing factor in causing abscess of the lung. If such is the case, it is not difficult to accept the theory of aspiration of infected material as the source of infection. That aspiration does occur has been shown by Myerson.[4] He made bronchoscopic examinations of 200 patients immediately following tonsillectomy and in 155 cases found blood and mucus in the trachea.

By experimentation with local anesthesia of the pillars, as done by Ochsner, it has been found that in satisfactory anesthesia the act of swallowing is abolished. This produces an immobility of the larynx, shown by bronchographic examination when a patient is given iodized oil and attempts to swallow. The oil can be seen passing into the larynx instead of into the esophagus. If, as has been shown, it is possible to abolish the reflexes of the throat when local anesthesia is used, it is also possible to establish the reflexes with general anesthesia; therefore, great care should be taken in the administration of the anesthetic not to allow the patient to be too greatly relaxed.

REPORT OF CASES

CASE 1.—A girl, a telephone operator, aged 19, reentered the New England Hospital for Women and Children on Sept. 3, 1910, with the diagnosis of gangrene of the lung. Two weeks previously she had been operated on in the department for diseases of the throat for removal of the tonsils and adenoids. After consultation with Dr. Frederic Shattuck the diagnosis was changed to abscess of the

* Submitted for publication, June 17, 1929.

1. Richardson, C. W.: Tonsillectomy with Consideration of Its Complications, Wash. M. Ann. **2**:28, 1912.

2. Moore, W. F.: Pulmonary Abscess: An Analysis of 202 Cases Following Operative Work About the Upper Respiratory Passages, J. A. M. A. **78**:1279 (April 29) 1922.

3. Ochsner, Alton: Bronchography Following the "Passive" Introduction of Contrast with Media Into the Tracheobronchial Tree, Wisconsin M. J. **25**:554 (Nov.) 1926.

4. Myerson, M. C.: Lung Abscess Following Tonsillectomy, Arch. Otolaryng. **1**:137 (Feb.) 1925.

lung following tonsillectomy. The illness had begun three days after the operation on the tonsils, with cough and raising of foul-smelling sputum a few days later.

At the time of entrance to the hospital the temperature was 101 F. and the pulse rate, 100, and there was a severe cough and a quantity of offensive sputum. Retention of the urine necessitated catheterization for three days after entrance. The patient's temperature was septic; it reached 104.6 F. on the seventh day and then gradually dropped. After the seventeenth day it did not go above 100 F.

At the time of admission, examination of the lung showed diminished breathing on the right side. The respiratory rate was 30; it was regular and there was no friction. There was tenderness on the right side below the clavicle. The resonance was deficient in the right apex, and dulness over the second and third ribs and some moist râles were noted. Sibilant râles were heard at base of the lungs. Only slight changes were observed during the next three weeks until there was a gradual clearing of all physical signs. Roentgenograms were not taken.

On laboratory examination, the blood showed: hemoglobin content, 75 per cent; red cells, 4,160,000, and white cells, 28,850. The sputum showed *Staphylococcus albus* in pure culture; the results of urinalysis were negative.

The patient was discharged from the hospital well on October 16, forty-four days after entrance.

Case 2.—A girl, aged 2½ years, reentered the New England Hospital for Women and Children on Dec. 1, 1927, two weeks after an operation on the tonsils and adenoids. The history was of general malaise, pain in the left shoulder and slight fever. She had been taken care of in the outpatient department previous to operation, with symptoms of underweight and a slight rise in temperature. There was a history of tonsillitis, enlarged glands and discharging ears.

On entrance to the hospital the patient appeared to be quite sick, and presented a listless, apathetic appearance. The wound in the throat was not entirely healed. The respirations were labored. The temperature was 102 F. The results of the examination of the chest were negative, and no physical signs were noted for two days, when an area of dulness developed at the angle of the left scapula. There was a loose sounding cough, and although no sputum was seen some was supposed to come away with the occasional coughing and vomiting spells.

The temperature rose as high as 104 F. on the sixth day, and from then for four weeks hung between 102 and 99 F. After the sixth week the temperature did not rise above normal.

The first roentgen report was consolidation of the left lung. The next showed the whole lung dense and the heart displaced to the right. The diagnosis was abscess of the lung. Frequent pictures and fluoroscopy showed the lung gradually clearing after about six weeks.

At the time of admission, the white blood cell count was 39,500. Two days later it was 44,200, and fifteen days after that it had reached 52,400. It was at this time that the patient was in the most serious condition. Two weeks later, when the next count was made, the temperature was on the downward trend and the white cell count had lowered to 20,000. The red cell count started at 5,550,000 and in four weeks was reduced to 2,980,000, with a hemoglobin content of 80 per cent, reducing to 70 per cent.

A smear from the larynx showed a few fungi resembling thrush. There was no reaction to tuberculin. The Wassermann reaction was negative. The urinalysis gave negative results.

The patient had a prolonged convalescence, extending over three months. At the time of writing, eleven months after the operation, the patient was in better physical condition than when she first went to the hospital, but was not entirely well as there was an occasional slight cough.

COMMENT

Considerable discussion has taken place as to the path of infection in abscess of the lung. Any interference with the protective reflexes may be a contributing cause to the inhalation of infected material. In the two cases reported, the first operation was done with the patient in the horizontal position without the use of a suction apparatus. If there was any dulling of the reflexes of the throat during the general anesthesia, infected material might easily have been inhaled.

The second case was that of an undernourished child, remarkable because of its lack of resisting force. The reflexes could have been overcome with a small amount of ether. This operation was done with the patient in the sitting position. In the past, it has been the custom in all the work done at this hospital to use the sitting position. Only an occasional operation is done with the patient in the recumbent position.

During the interval of seventeen years between the two cases reported here, about 4,000 patients have been operated on in the department for diseases of the nose and throat. It is impossible to say that no other abscess of the lung has resulted, but great effort has been made to do follow-up work on every patient. Each patient reports at the outpatient department within ten days after the operation and is told to return again if she is not feeling well, or to send in a call so that she may be attended at home by an intern.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

PLASTIC SURGERY

J. EASTMAN SHEEHAN, M.D.
NEW YORK

No sooner had it been demonstrated that the use of acriflavine hydrochloride in acetone solution as a skin dressing immediately antecedent to surgical incision afforded increased security against subsequent infections, the origin of which was in the skin itself, than new experiments indicated that there was still a higher degree of immunity to be had by substituting a mixture of methylene blue (methylthionine chloride, U. S. P.) and gentian violet. Since in all these procedures for reorganization and replacement infection is peculiarly objectionable, because of its liability to produce new disfigurements, measures directed toward its prevention are of essential interest. The order of the progress made in this field is significant. First, there was almost implicit dependence on the efficacy of iodine. Then it was shown that several forms of infection-producing germ life were immune not only to iodine but to other solutions. And now the experiments conducted by Kingsley Roberts [1] take into account infection-producing material carried to the surface from the sweat glands and sebaceous glands during the term of the operation itself, so that there is additional merit in a wash that will continue to be effective for two and a half hours. In every branch of science, almost, some new wonder has been evolved from these dyes; that they should have given us an almost perfect and inexpensive antiseptic, which is nontoxic, nonirritant and one which neither interferes with the function of the skin nor retards the process of restoration, is surely another score to their credit.

One notes with interest, also, that there continues to be intelligent experiment in methods of treatment for devastating burns. For the tannic acid treatments it is claimed that, at the least, the leather-like fixation tends to prevent that absorption of proteins from the injured cells which accounts for so much distress, not to say danger. It will

* Submitted for publication, Sept. 7, 1929.

1. Roberts, Kingsley: Value of Methylene Blue-Gentian Violet 5 Per Cent in Pre-Operation Skin Preparation, Ann. Surg. **89**:183 (Feb.) 1929.

always be doubtful whether skin substance thus structurally altered can continue, in all situations and for an indefinite time, to discharge the functions demanded of it. The mere fact of the change creates at least a speculative interest in the ultimate need of reparative surgery.[2] Similarly, if in an opposite sense, one must be attracted to the recent experiments in treating burn areas with normal horse serum, for which it is claimed that, besides its healing influence, its use admits of, and indeed promotes, renewal of the cell tissues that had been destroyed.[3]

From these considerations it is but a step to the sound advice given by Blair, out of his long and varied experience, that when it is obvious that there must be skin replacement to obviate the evils of contractures, it is best, other things being equal, to proceed early rather than to await developments the progress of which can be infallibly foreseen.[4]

In the additions to the knowledge regarding skin grafting, there are two contributions worthy of more than passing mention. Continuing his research experiments, Staige Davis[5] has demonstrated the possibilities in the spread of epithelization outward from innumerable islands in the form of minute pinch grafts spread over a large denuded area. It may be said, indeed, that in practice the recourse to this method is not likely to be frequent; there is hardly any defect that cannot be covered by the thin epidermic shaving called the Thiersch graft, which "takes" in practically 100 per cent of the cases, and the choice, if any, between the complete epidermic covering and one that is mottled by the thicker pinch grafts, will not be for the latter. But that is beside the point. The value of these experiments is in the demonstration of the aid given by nature in affording cover, the importance of which will be realized by any one who has seen, for example, the formation of a lining within a cavity from which the lower mandible had been removed and while a prosthesis was in place.

On the practical side, notably as an indication of the confidence that comes from familiarity and experience, is the development in connection with the use of a graft which in thickness as well as, necessarily, in physiologic attributes is half way between the merely epidermic graft and the full-thickness free graft. There are some situations in which it is undesirable that any part of the corium should adhere to the epidermic shaving; e. g., in the lining of an eye socket, in which hairs and exudations are to be apprehended if any of the subpapillary layers

2. Montgomery, A. H.: Tannic Acid Treatment of Burns in Children, Surg. Gynec. Obst. **48**:277 (Feb.) 1929.

3. Monteith, S. R., and Clock, R. O.: Treatment of Burns with Normal Horse Serum, J. A. M. A. **92**:1173 (April 6) 1929.

4. Koch, S. L., and Kanavel, A. B.: Contractures Due to Burns, J. A. M. A. **92**:277 (Jan. 26) 1929.

5. Davis, J. Staige: Ann. Surg. **89**:902 (June) 1929.

are transplanted. But there are, obviously, situations in which the main advantage of the epidermic graft consists in the fact that the area to be covered is too large for one to venture on with a full-thickness free graft. Restoration of the full-thickness graft is as long and as complex physiologically as that of the Thiersch graft is rapid and simple. Manifestly, if a half-thickness graft can be utilized to cover large defects, even up to 100 square inches, as dependably as the Thiersch graft, with restoration in a few days instead of three weeks and with infinitely less disturbance of the donor area, there is an immense addition to the munition of the reparative surgeon. Blair's paper [6] on the subject is therefore to be rated as embodying one of the most notable advances of recent years, an advance which has only been made possible by the recent concentration on reparative surgery as a special department.

It may be useful to note in this connection that replacement is now seen to serve a most useful purpose in many instances in which applications of radium and the x-ray have caused misadventures.[7] Sometimes one has to deal with merely negative consequences, as when the irradiation has not proved successful (e. g., the occurrence of angiomas, etc.) and as a result the surrounding tissues have been devitalized by the action of the emanations. At other times burns, more or less extensive, or scars, more or less destructive of function, compel intervention. It is worth a great deal to know that such intervention can be accounted, in the main, adequately effective.

COMMENT

Of the individual cases reported, one is interesting in the field of general surgery in that it stresses the advantage of closing the skin wound over the fracture of a bone, the advantage gained depending on the creation of secondary defect areas on both sides, to be covered by replacements.[8] In another, involving the splicing of a tendon, in which a section of vein was used to enclose the transplant, the vein is reported as having become indistinguishably incorporated with the tendon, not as a sheath but as a living part of it.[9] Finally, since the report has attracted some attention, I may be allowed to mention the restoration of a thumb nail by means of a graft from the nail of the other thumb.[10]

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6. Blair, Vilray P., and Brown, John: Use and Uses of Large Split Skin Grafts of Intermediate Thickness, Surg. Gynec. Obst. **49**:82 (July) 1929.

7. Footnote 6. J. Eastman Sheehan: X-Ray and Radium Burns, Laryngoscope **38**:612 (Sept.) 1928.

8. Cannaday, John E.: Value of Closing Compound Fractures by Skin Plastics, Ann. Surg. **89**:597 (April) 1929.

9. Auchincloss, Hugh: Tendon Transplantation, Ann. Surg. **89**:145 (Jan.) 1929.

10. Sheehan, J. Eastman: Restoration of Thumb Nail, J. A. M. A. **92**:1253 (April 13) 1929.

Miscellany

REPRODUCTION OF PHOTOMICROGRAPHS

The reproduction of photomicrographs in this journal is limited by the available space on the page.

The column width is 4¼ inches. All cuts have a ⅛ inch shoulder on each side to permit of the metal being fastened to the wood block; hence, if consistent with the material to be shown, illustrations are reduced to column width—that is, to 4 inches. Larger cuts not only cost more but necessitate additional expense in arranging makeup. When necessary or especially desirable, larger reproductions can be produced, but only in very exceptional instances are cuts made more than 5½ inches wide.

When photomicrographs more than 5½ inches wide are submitted, authors should decide whether they prefer to have only the significant portion of the illustration reproduced full size or to have the entire picture shown with reduction. Notation of the choice should accompany the manuscript; in case choice is not indicated, manuscript editors will use their own judgment as to the procedure to be followed.

Abstracts from Current Literature

Ear

POSTANGINOUS PYEMIA. RICHARD WALDAPFEL, Ztschr. f. Hals-, Nasen- u. Ohrenh. **23**:178 (June) 1929.

Postanginous complications appear: (1) as submucous complications, for example, peritonsillar abscess; (2) as disease of the lymph glands without injury of the wall of the pharynx, i. e., (*a*) a swelling or excessive enlargement or (*b*) an abscess with possible propagation toward the mediastinum, the base of the skull or the blood vessels; (3) as a direct penetration through the wall of the pharynx, namely, a parapharyngeal abscess with the possibilities mentioned under 2 *b*, and (4) as a direct transmission of the microbes from the tonsil to the blood stream and their rapid distribution through the whole system. Viewed from the standpoint of therapy, cases of postanginous pyemia can be classified into two groups. Cases of the first group, with a tremendously rapid course, show no swelling of the neck and present the picture of a fulminating sepsis. It is entirely unknown whether a greater virulence of the microbes or a diminished resistance of the body, especially of the glandular apparatus, is to be taken into consideration. Cases of the second group are more localized, and show formation of abscess and swelling of the outer part of the neck. The first group is characterized by an infection of the blood-current, originating in the tonsil. The prognosis is bad and the therapy thus far futile. For the second group, surgical intervention is indicated. The pharynx muscles and the pharyngeal fascia can be reached (1) from the anterior and posterior palatine arch or through the tonsil, and (2) from the outside under the digastric muscle, by penetrating between the lower jaw and the pharyngeal wall. If the pharyngeal wall and the fascia have been penetrated by the inflammatory process, the second route is the more successful one, because it meets the suppurative process and does not run after it and because it makes drainage possible at the lowest point, if intervention has come in time. This method with possibly a preliminary preventive mediastinotomy in the neck after Marschik and preventive exclusion of the mediastinum of the thorax, is used in the Vienna clinic in the majority of cases. The resection of the thrombosed veins encountered in the operation is, naturally, always carried out. Local anesthesia is employed without resort to the dangerous plexus anesthesia. Schlemmer gave the following indications for mediastinotomy in acute septic processes of Waldeyer's ring: (1) in the case of certain local symptoms in the pharynx; (2) if there is plastic inflammation in the region of the soft parts of the neck on the diseased side with considerable tenderness on pressure along the large vessels of the neck as far as the clavicle; (3) if inflammatory swelling progresses quickly in the course of a few hours; (4) if there is high fever (of continuous or intermittent type); (5) if the pulse is frequent and soft; (6) if unrest or slight stupor is present, and (7) if intravenous use of serums is not of prompt effect or of no effect. Forty-three cases were investigated, in two of which microscopic serial sections of the tonsils and surroundings were available. In the serial sections, a primary thrombosis of the veins of the tonsil was established, but a primary thrombosis of the internal jugular vein was excluded. This vein itself was free from thrombus in its upper portion. The subsidence of the thrombus at the upper pole of the abscess and of the tonsil speaks against a retrograde continuation of the thrombus. Yet retrograde thrombi can occur. This is proved in a case in which a thrombosis continued almost to the other tonsil. Thrombosis was not invariably accompanied by an invasion of microbes. A large number of minute, small and large veins were found thrombosed, but an invasion of microbes took place in only a few spots. This invasion did not take place in the tonsil itself, but outside, either from the abscess or from the lymph channels

of the parapharyngeal space. In the great majority of cases, abscesses were found. Direct infection of the blood current from the tonsils without the formation of abscess occurred only in a small percentage of cases. Four cases showed that an infection can take place as the result of a suppurating gland. When the formation of abscesses could be controlled, a cure resulted; when not, the outcome was fatal. In the first instance cure resulted even when no vein was ligated or excised; in the latter instance, the result was fatal even after ligation and excision of veins. The abscess near the tonsil must be viewed as a focus of the sepsis in the sense of Schottmueller. Schottmueller held that an increase of pathogenic germs does not occur in the blood current in any microbic infectious disease in man. The same holds true in most severe septic diseases; the pathogenic microbes of the blood always issue from a local focus. Consequently, the therapy consists in the elimination of this focus. All interference with the venous system is of secondary importance. The opening of the parapharyngeal space, after possibly a preliminary preventive mediastinotomy of the neck and preventive exclusion of the mediastinum, is the method of choice. This method also makes the lymph vessels of the parapharyngeal space accessible, if there is no pronounced formation of an abscess present, and makes possible the prevention of an extension of the process downward. Possibly, thrombosed veins are resected, if they can be recognized as such and if they are accessible. The time for intervention varies. It is better to intervene too soon than too late. One should not wait until pus can be demonstrated in the sheaths of the vessel. The author did not encounter the picture of postanginous pyemia in children.

AMBERG, Detroit.

Miscellaneous

PATHOGENESIS OF SEPSIS AFTER ANGINA. HISTOLOGIC OBSERVATIONS IN THE MATERIAL OF TWENTY-SIX PATIENTS OF PROFESSOR CLAUS, BERLIN. S. BURCHARDT, Ztschr. f. Hals-, Nasen- u. Ohrenh. **23**:97 (March 23) 1929.

The author publishes a report based on observations in eleven necropsies, in ten cases in which the patients were cured and in five cases (bordering on sepsis) in which especially enucleated tonsils were available. In these five cases, she noticed perivascular infiltration. The lumen of the vessels was free. The lymph spaces were filled with cells and cocci. All patients showed swelling of the regional lymphatic glands. This perivascular infiltration was a common occurrence. It always started in the tonsil or tonsillar bed, and could be followed, in the eleven necropsies, macroscopically and microscopically, to the large veins. Here the lymph glands were always infiltrated and mostly matted together. In six cases, contact infection was found between lymph nodules and the venae faciales communes or jugulares, in the lumen of which a suppurative thrombophlebitis was encountered immediately adjacent to the diseased lymph glands. The walls of the vessels showed inflammatory disintegration in all layers. The other five cases showed, plainly, adhesions between the infiltrated perivascular tissue and the walls of the vessels, partly to the bed of the tonsil. In one case, a destructive inflammation could be observed reaching to the media, whereas the intima was intact and the lumen empty. Uffenorde's theory is strengthened by the observation that the pathways of the tonsil are filled downward with cells, thrombotic masses, cocci, etc., especially since no proof could be adduced for a purely hematogenous process of propagation, macroscopically or microscopically. Isolated lesions of the intima were never observed. The main point in postanginous complications seems to be the formation of a more or less pronounced phlegmon which propagates perivascularly. The lymph channels are undoubtedly involved. If the perivascular filtrates reach the blood vessels, they do so at any site, either near the original focus or deeper, affecting the venae faciales communes or jugulares. Whether or not a thrombophlebitis develops is of secondary importance so far as the genesis is concerned. The divergence of opinions of Fraenkel and Uffenorde can be explained in this manner.

AMBERG, Detroit.

Society Transactions

AUSTRIAN OTOLOGIC SOCIETY

December 17, 1928

E. URBANTSCHITSCH, M.D., *Chairman*

DR. E. URBANTSCHITSCH delivered an address in memory of Professor Kreidl.

ABSCESS OF TEMPORAL LOBE FOLLOWING ACUTE OTITIS MEDIA. By DR. O. MAYER.

Dr. Mayer presented a case in which abscess of the temporal lobe developed after acute otitis media. In spite of the fact that encephalomalacia rubra developed following the opening of the abscess, the patient recovered after continuous tamponade with iodoform gauze.

DISCUSSION

DR. LEIDLER: In two cases I used continuous tamponade. One of the patients recovered; the other one died.

DR. H. NEUMANN: The method of Albrecht, Feuchtinger and Leidler requires not only tamponade for the cavity of the abscess, but also removal of the inflamed cerebral substance. This procedure is then followed by tamponade. The method is valuable for abscesses of the convexity; however, for abscesses on the base of the brain the procedure of Lemaitre seems preferable. It must be admitted that even this method does not prevent encephalitis, which in the healing of a cerebral abscess is of even greater significance than meningitis.

DR. LEIDLER again described the method of continuous tamponade in cerebral abscesses.

DR. O. MAYER: Tamponade for cerebral abscesses is really not an entirely new procedure. The only new feature is that the tampon remains in for several weeks. The value of Lemaitre's method must be proved by future results.

OTITIS MEDIA IN PNEUMONIA DURING CHILDHOOD. By DR. Z. HOFMANN.

Dr. Hofmann presented a statistical clinical consideration of a large material.

DISCUSSION

DR. RUTTIN: Whenever, in cases in which otitis media appears as a prodrome of pneumonia, high fever exists and the tympanic membrane is not macerated but is still transparent, the cause of the feverish condition is usually not in the ear but in the lungs.

DR. O. MAYER: Otitis media in pneumonia is usually mild. However, in some cases early operation becomes necessary.

DR. H. NEUMANN: In acute otitis, a certain congruence exists between the condition of the tympanic membrane, the pains and the temperature. If this cannot be observed, one should search for other causes of the symptoms.

DR. LEIDLER: We have no definite knowledge concerning the causes of fever in acute otitis media.

DR. FREY: I am not convinced of the importance of avoiding a few paracenteses, since they are entirely harmless.

DR. RUTTIN: If a paracentesis is performed, the healing process requires a longer time, because following paracentesis a mixed infection with staphylococci usually develops. The pains in otitis in pneumonia occasionally are due to vesicles on the tympanic membrane.

DR. H. NEUMANN: Paracentesis should not be performed in all cases, but only when there are indications for this procedure.

DR. H. FREY: I also am not in favor of paracentesis in those instances in which the tympanic membrane has retained its sheen. The conditions are entirely different in cases of secretory catarrh of the middle ear. Neumann considers this catarrh as a mild form of otitis media. However, transitional conditions between otitis media and secretory catarrh are rarely observed, and consequently this assumption is not justified.

DR. RUTTIN: I saw two cases in which acute otitis media with sinus thrombosis developed after a paracentesis for secretory catarrh.

DR. FORSCHNER: I have observed a case of secretory catarrh that developed into one of acute otitis media as the result of paracentesis.

DR. HOFMANN: In otitis media in pneumonia, the tympanic membrane occasionally shows serious changes, especially in cases in which high temperatures may be traced to the pneumonia. In these instances, paracentesis does not represent an entirely harmless procedure.

January 28, 1929

C. BONDY, M.D., *Chairman*

DR. BONDY presented a case of healed fistula of the semicircular canals.

DR. RUTTIN showed two cases of otitis media caused by *Diplococcus lanceolatus*. One patient recovered without operation, in spite of the fact that during roentgenoscopy destruction of bone in the mastoid process could be observed. The other patient presented meningitis at the end of the second week.

DR. RUTTIN described a case of acute otitis media with mastoiditis. Following the operation, a coronal swelling and suppuration of the scalp developed and Korsakoff's psychosis became manifest.

DR. RUTTIN showed the specimens in a case in which an acute otitis media had led to meningitis in eight days. The pathogenic micro-organism was *Streptococcus pyogenes*.

DR. SCHLANDER showed the microscopic specimens in a case in which a suppuration of the middle ear entered the carotid canal and caused erosion of the carotid artery.

DR. BARWICH discussed new pathogenic agents of otitis media, meningitis and pneumonia.

February 25, 1929

MENINGITIS AND PHLEBITIS OF THE CAVERNOUS SINUS, FOLLOWING SUPPURATIVE DEGENERATION OF A CHOLESTEATOMA. DR. RUTTIN.

Dr. Ruttin showed the specimens in a case in which meningitis and phlebitis of the cavernous sinus, with their typical symptoms, followed an acute suppurative degeneration of a cholesteatoma.

FIBRINOUS OTITIS MEDIA. DR. RUTTIN.

Dr. Ruttin presented a case of fibrinous otitis that was characteristic, the perforation being closed again and again by fibrinous masses.

DISCUSSION

DR. EISINGER: What type of bacteria was present. In a similar case, that came under my observation, *Streptococcus hemolyticus* was found.

DR. RUTTIN: In one instance, staphylococci were detected and in the other case, streptococci.

DR. SCHLANDER: Was diphtheria found?

DR. RUTTIN: Never.

SCLEROSIS OF THE PNEUMATIC SYSTEM. DR. EISINGER and DR. E. C. MAYER.

Roentgenograms were shown which proved that parts of the pneumatic system may lose the original structure and become sclerotic.

ABSCESS OF THE TEMPORAL LOBE FOLLOWING A RADICAL OPERATION. By DR. E. URBANTSCHITSCH.

Two years after a radical operation, an abscess developed in the temporal lobe. The clinical manifestations, however, indicated an abscess of the cerebellum. It could not be detected with the aid of puncture of the brain.

DR. NEUMANN: In this case, the cerebral abscess was present at the time of the radical operation; otherwise, this would be one of the rare occasions on which the radical operation did not protect the patient against intracranial complications.

DR. URBANTSCHITSCH: I assume that the case in question concerned an old cerebral abscess.

EXCLUSION OF THE LABYRINTH BY A CHOLESTEATOMA. By DR. NEUMANN.

As a sequel of a cholesteatoma in a child, exclusion of the labyrinth developed. During the operation, the cholesteatoma was found far in the inner ear. It was decided therefore to perform an operation on the labyrinth, although, apart from the exclusion of the inner ear, there were no other clinical aspects giving indications for an operation on the labyrinth.

VIENNA LARYNGORHINOLOGIC SOCIETY

July 3, 1928

DR. HAJEK, *Chairman*

SEVERE EPISTAXIS FROM HEMANGIOMA OF NASAL MUCOUS MEMBRANE. By DR. RIENZNER.

As a result of a hemangioma of the nasal mucous membrane, an epistaxis developed which endangered life.

DISCUSSION

DR. KREN discussed the differential diagnosis of the various forms of nevus and pointed out that the condition in the case in question was nevus vasculosus.

DR. M. WEIL: I have observed a similar case.

DR. MARSCHIK: The small, frequently overlooked angioma of the raphé palati often leads to occult hemorrhages, and these are diagnosed as pulmonary hemoptysis.

CHRONIC EDEMA (IDIOPATHIC ELEPHANTIASIS) OF THE MUCOUS MEMBRANE OF THE NOSE, PHARYNX AND LARYNX. By DR. RIENZNER.

A cause for this condition could not be detected in the case presented, and various treatments were of no avail.

SYNECHIA OF VOCAL CORDS FOLLOWING CURETTAGE OF LARYNX. By DR. RIENZNER.

Dr. Rienzner presented a case in which, following curettage of the larynx on account of productive laryngeal tuberculosis, a synechia of the vocal cords

developed. An operation was performed according to Haslinger's method (insertion of a silver platelet between the vocal cords). It was surprising that around the platelet granulations developed, so that even seven weeks after the operation the platelet could not be removed.

DISCUSSION

DR. HASLINGER: I never have observed an abundant development of granulations. However, I have performed the operation on a larynx that previously had been affected with tuberculosis.

DR. MAYER: I operate only when there are no signs of previous tuberculosis.

ADENOID HYPERPLASIA WITH LYMPHATIC LEUKEMIA. By DR. BUSTIN.

A case of adenoid hyperplasia especially of the lingual tonsil with lymphatic leukemia was presented.

DISCUSSION

DR. HAJEK: Was anything found in the nasal conchae?

DR. BUSTIN: Nasal examination gave negative results.

UNILATERAL RHINITIS ATROPHIA FOETIDA. By DR. STERNBERG.

This case of ozena occurred in a child, aged 3.

A CASE FOR DIAGNOSIS. By DR. WEIL.

A snow-white, thin, irremovable coating was found on the right tonsil, with painful swelling of the submaxillary glands. The tentative diagnosis was pneumococcus angina.

DISCUSSION

DR. HAJEK: The condition may be lymphosarcoma.

CARCINOMA OF THE TONSIL. By DR. HIRSCH.

A carcinoma of the tonsil had spread to the base of the tongue and had formed metastases in the lymph nodes. Six years after the operation, the patient had not had a relapse.

EMPYEMA OF THE ETHMOID BONE. By DR. HIRSCH.

Dr. Hirsch showed the specimens of a circumscribed empyema of the cells of the ethmoid bone. The cells were pushed forward into the middle concha and showed marked dilatation.

ANESTHESIA WITH A HYPNOTIC PREPARATION. By DR. F. J. MAYER.

Dr. Mayer discussed his experiences with a hypnotic preparation as an anesthetic that is administered by intravenous injection. The disadvantage of this narcotic agent is the long duration of the sleep after the operation. This kind of narcosis, therefore, is advisable only for those operations that require much time or for those that have a choking effect.

DISCUSSION

DR. RIENZNER: Excitement may be observed in the patient following the injection of the hypnotic. It is impossible to determine the duration of the sleep that follows.

DR. FORSCHNER: I have observed no favorable results with the preparation.

DR. MAYER: In spite of its disadvantages, the hypnotic is of practical value.

SYMMETRIC TUMORS OF THE TYMPANUM. By DR. MENZEL.

It was considered possible that this case of symmetric tumors on the fold of the mucous membrane of the tympanum was one of laryngocele.

CARCINOMA OF THE HYPOPHARYNX. By DR. HASSLINGER.

A resection of the pharynx was done. The larynx was spared.

MEDIASTINITIS AND EMPHYSEMA OF THROAT FOLLOWING PERFORATION OF THE TRACHEA AND THE ESOPHAGUS BY PUS FROM A PERIBRONCHIAL TUBERCULOUS AND SUPPURATIVE LYMPH NODE. By DR. HASSLINGER.

Dr. Hasslinger showed specimens from a case.

PHLEGMON OF THE THROAT FOLLOWING TONSILLECTOMY. By DR. WALDAPFEL.

An operation was performed from the outside, and it brought temporary improvement. Later, the inflammation spread toward the mediastinum. A second operation was performed and drainage was done according to Seiffert's method. Healing followed.

A CASE FOR DIAGNOSIS. By DR. GROSSMANN.

Microscopic specimens were shown the microscopic examination of which did not definitely establish whether the case in question was one of lymphogranulomatosis, tuberculosis or syphilis of the upper air passages.

November 6, 1928

DR. PANZER, *Chairman*

CARCINOMA OF THE ETHMOID BONE. By DR. ZEIDLER.

Radical operation and radium treatment led to recovery.

ROENTGEN INJURY OF THE LARYNX. By DR. ZEIDLER.

The injury became manifest as a temporary unilateral perichondritis of the larynx.

A CASE FOR DIAGNOSIS. By DR. STERNBERG.

An involvement of the mucous membrane at first presented the clinical aspects of a pneumococcic infection. At the time of the meeting, it appeared to be pemphigus.

DISCUSSION

DR. PANZER: I have observed a similar case. I also have seen cases that took a malignant course.

DR. GLAS: I consider the condition pemphigus.

DR. MENZEL: The prognosis is unfavorable in cases in which pemphigus of the mucous membrane occurs simultaneously with efflorescence of the skin.

DR. STERNBERG: The differential diagnosis between pemphigus and erythema multiforme is difficult.

SULCUS GLOTTIDEUS. By DR. STERNBERG.

A longitudinal furrow was observed on the left vocal cord.

DISCUSSION

DR. HOFER: These furrows are always congenital, and in extreme cases they even lead to a doubling of the vocal cord.

DR. STERNBERG: In this case, the hoarseness is not caused by the longitudinal furrow but rather by a laryngotracheitis.

EXOPHTHALMIC GOITER CAUSED BY AN IODOFORM TAMPON. By DR. SUCHANEK.

Following a tonsillectomy, a tampon had remained in a tonsillar niche for twenty-four hours. Exophthalmic goiter developed.

DISCUSSION

DR. HOFER: How long after the operation was it before the goiter appeared? I question the connection with the iodoform tampon.

DR. HEINDL: The goiter might have been caused by the operative trauma.

DR. HUTTER, DR. STERN and DR. MARSCHIK were of the same opinion as Dr. Heindl.

DR. SUCHANEK: I do not think that the operative trauma had any influence.

CARCINOMA OF THE MOUTH. By DR. KOLISCH.

The patient recovered, following radical operation with a simultaneous plastic operation, according to Esser's method.

TONSILLAR CALCULUS. By DR. HEINDL.

The tonsillar calculus had for a long time simulated a disintegrating carcinoma of the tonsil.

PLASMOCYTOMA. By DR. WEIL.

Dr. Weil again presented the case shown in the last session. It finally was found to be a plasmocytoma.

POSTOPERATIVE BILATERAL POSTICUS PARALYSIS. By DR. WEIL.

Dr. Weil presented a patient who after repeated thyroidectomies developed a bilateral posticus paralysis. In order to relieve the dyspnea, excision of the vocal cord was performed.

DISCUSSION

DR. HOFER: In cases of median stenosis, the best results have been gained with a persistent tracheal fistula.

DR. KOFLER: Tracheostomy gives the best effects. However, in certain cases I would proceed in a different manner. In a tracheotomized patient, I would perform a deep cauterization, one starting far away from the edge of the labium vocale and extending over its entire length. Undoubtedly, this procedure impairs the voice.

DR. SUCHANEK: I have found that the epithelized fistula of long duration is satisfactory.

DR. WEIL: I consider the excision of the vocal cords as more advantageous.

BASAL CELL CARCINOMA OF THE EPIPHARYNX. By DR. GLAS.

A basal cell carcinoma of the epipharynx was removed by endonasal operation.

PEDUNCULATED CARCINOMA OF THE BASE OF THE TONGUE. By DR. GLAS.

The carcinoma was removed by means of a galvanocautery loop.

HEMANGIOMA OF THE MIDDLE CONCHA OF THE NOSE. By DR. GLAS.

Histologic specimens were shown.

A NEW BRONCHO-ESOPHAGOSCOPIC MODEL AND SEVERAL NEW LARYNGOLOGIC MODELS. By DR. HASSLINGER.

A Case for Diagnosis. By Dr. Pordes.

Submucous swellings of the larynx and the pharynx became manifest after roentgen irradiation of the throat. It was questioned whether this condition was due to roentgen injury or to tuberculosis.

Ranula Following Tonsillectomy. By Dr. Hofmann.

The ranula developed in the scar of a tonsillectomy.

DISCUSSION

Dr. Glas: The visible cyst, perhaps, can be traced to the lymph nodes in the region of the tonsillar capsule.

Dr. Hutter: I do not think that in this case the visible cyst originated in the submaxillary glands.

Dr. Kellner: I would bear in mind the hypertrophic salivary glands that are found occasionally as appendixes of the tonsil; the cyst may have developed from this origin.

Dr. Hofmann: In this instance, the etiology of the cyst is not clear.

Pseudotumor of the Nasal Cavity. By Dr. Menzel.

The pseudotumor became manifest as a splinter of a congenital prominentia tubaria pharyngis.

DISCUSSION

Dr. Ruttin agreed with Dr. Menzel as to the diagnosis.

The Forcing Out of a Paraffin Prosthesis in the Septum. By Dr. Menzel.

Nine years after the introduction of hard paraffin into the septum on account of ozena, the prosthesis was forced out.

December 4, 1928

Dr. Hajek, *Chairman*

Plasmocytome. By Dr. Harmer.

Dr. Harmer showed the specimens of a bronchial tumor that proved to be a plasmocytoma of the left bronchus. The case had been presented at a former session.

Inflammatory Destruction of Ethmoid Bone and Perforation into Cranial Cavity. By Dr. Cemach.

Inflammation of the ethmoid bone, without perceivable suppuration and without a specific factor being involved, had led to the complete destruction of the ethmoid bone and to perforation into the cranial cavity. It was also noted in this case that when cocaine or epinephrine hydrochloride was administered, a swelling of the nasal mucous membrane developed.

DISCUSSION

Dr. Kofler: Repeated inunctions with cocaine effect a swelling of the nasal mucous membrane. However, if it is applied only once, with a brush, the swelling disappears.

Dr. Sternberg: It is not absolutely necessary that while the cocaine acts on the nasal mucous membrane the swelling should disappear.

DR. TSCHIASSNY: If the mucous membrane constantly fails to react following the application of cocaine, a specific infiltration may be suspected.

LOSS OF VOICE FOLLOWING EXTIRPATION OF THE LARYNX. By DR. MARSCHIK and DR. FROESCHELS.

Following extirpation of the larynx, the patient's pharyngeal voice would not function. The patient finally discovered that inspiration of air into the esophagus made it possible to use the pharyngeal voice.

DISCUSSION

DR. MARSCHIK: With the aid of esophageal inspiration and expiration, the pharyngeal voice should be more extensively cultivated.

DR. FROESCHELS showed roentgenograms in which the esophageal breathing was clearly visible.

DR. STERN: Esophageal respiration is not a natural process, for in most instances in which the patients acquire their pseudovoice spontaneously, the air reservoir is to be found in the upper segment of the stomach.

DR. MARSCHIK: The local recidivation of carcinoma often is the cause of a failure of the pharyngeal voice to appear. However, in this particular case, that was not the reason.

BILATERAL POSTICUS PARESIS. By DR. MARSCHIK.

It could not be definitely determined whether the bilateral posticus paresis was organic or functional. The opinion was expressed that in a case of bilateral posticus paresis tracheotomy should occasionally be performed even when the paresis is of a functional nature.

DISCUSSION

DR. MENZEL: I saw two patients with functional stenosis of the larynx. One of them had to be tracheotomized repeatedly.

DR. HOFER: These cases may be considered as spasms rather than as posticus paresis.

DR. HEINDL: I have seen a patient with aphonia and dyspnea spastica.

DR. HUTTER and DR. TSCHIASSNY mentioned similar cases. DR. MARSCHIK stressed the difficulty of a differential diagnosis between spasm and paresis.

MEDIASTINITIS AND GENERAL INFECTION AFTER SWALLOWING OF A NEEDLE. By DR. RIENZNER.

The needle was removed seven days after the patient had swallowed it. The general infection and the mediastinitis had already developed. The mediastinitis was counteracted by bilateral cervical mediastinotomy.

EDEMA OF LARYNX AND PERIESOPHAGEAL ABSCESS AFTER SWALLOWING A BONE. By DR. RIENZNER.

Three days after the swallowing of a bone, there developed, at the level of the cricoid cartilage, edema of the larynx with a periesophageal abscess. Following tracheotomy and posterior mediastinotomy, the patient recovered.

SUBMUCOUS ABSCESS OF THE ESOPHAGUS AS A RESULT OF SWALLOWING A BONE. TWO CASES. By DR. RIENZNER.

In the first case, seven days after the patient had swallowed a bone, a submucous abscess developed beneath the mouth of the esophagus. It was emptied from the inside. In the second case, a submucous abscess of the esophagus resulted from the swallowing of a bone.

CARCINOMA OF THE LOWER LIP AND OF THE RIGHT VOCAL CORD. By DR. FORSCHNER.

This was a case of benign carcinosis.

MUCOCELE OF THE FRONTAL SINUS AND OF THE LABYRINTH OF THE ETHMOID BONE. By DR. HAJET.

Following an endonasal operation, the patient recovered.

CARCINOMA OF THE RIGHT FRONTAL SINUS. By DR. HAJEK.

Dr. Hajek presented a case of primary carcinoma of the frontal sinus with perforation and with inflammatory manifestations.

HYPERNEPHROMA OF THE FRONTAL SINUS. By DR. HAJEK.

Dr. Ruttin mentioned, in comment, a case of carcinoma of the frontal sinus.

CARCINOMA OF THE WALL OF THE HYPOPHARYNX. By DR. HAJEK.

Resection of the pharynx was done. The larynx was spared.

A CASE FOR DIAGNOSIS. By DR. BENESI.

Dr. Benesi again presented the previously demonstrated case of lymphogranulomatosis of tonsil and throat. He remarked that even yet the diagnosis was not completely clear.

GENERAL LIPOIDOSIS OF THE SKIN AND OF THE MUCOUS MEMBRANES OF THE RESPIRATORY PASSAGES. By DR. WIETHE.

The case was congenital and familial on the basis of a latent diabetic disturbance of the metabolism.

DISCUSSION

Dr. Urbach discussed the differential diagnosis of this disease.

DR. KOFLER: I have noted similar cases, but I have always incorrectly diagnosed them as cases of papillomatosis of the larynx.

PERITONSILLAR ABSCESS LEADING TO THROMBOSIS OF THE JUGULAR VEIN AND SEPSIS. By DR. BUSTIN.

Histologic specimens were shown.

DISCUSSION

DR. MARSCHIK: I question the effectiveness of a ligation of the jugular vein in cases of septic processes in the tonsils. If the ligation is performed, it should be done early and with simultaneous exposure of the parapharyngeum and with the vein having been made impermeable to the surroundings.

DR. RUTTIN: I consider the ligation of the jugular vein valuable whenever the infection originates in those lymph nodes that are near the jugular vein. However, success will be less certain if the general infection originates in a septic angina, not in a lymph node, but directly in small vessels, whether they are thrombosed or not, by way of the jugular vein.

DR. WALDAPFEL: The infection of the jugular vein by way of a lymph node is not the rule. Usually an abscess may be found in the parapharyngeal space. On the operative treatment of this abscess depends the fate of the patient.

DR. HIRSCH: Cases of sepsis may occur after tonsillectomy.

DR. BUSTIN: In the case reported by me, the jugular vein was entirely free from infection.

DEVIATION OF THE SEPTUM AND SADDLE NOSE, DUE TO A FALL ON THE FACE. By DR. TSCHIASSNY.

Resection of the septum was performed, and a piece of vomer with "Krista," 2.5 cm. in length, was implanted subcutaneously.

ARTIFICIAL OBSTRUCTIONS OF RESPIRATORY TRACT. TWO CASES. By DR. KISSMANN.

Dr. Kissmann showed the nut of a screw which he had removed from the rhinopharynx of a child aged 18 months; also a rhinolite that had been in the nose of a patient for twenty years.

BILATERAL SYMMETRIC ANOMALY OF THE NASAL DUCT. By DR. GUIST.

Directory of Otolaryngologic and Ophthalmologic Societies *

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. William V. Mullin, Cleveland Clinic, Cleveland.
Secretary: Dr. Gabriel Tucker, Bronchoscopic Clinic, University Hospital, Philadelphia.
Place: Detroit. Time: June 23-27, 1930.

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AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY

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Secretary: Dr. R. L. Loughran, 33 E. 63d St., New York.
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AMERICAN OTOLOGICAL SOCIETY

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Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

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Place: Atlantic City, N. J. Time: Oct. 21-25, 1929.

LOCAL

LOS ANGELES COUNTY MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Harold S. Muckleston, 6422 Hollywood Blvd., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Place: California Hospital, assembly room. Time: 8 p. m., first Monday of each month.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

THE COLORADO OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Frank L. Dennis, Ferguson Bldg., Colorado Springs.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION ON OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Dr. Louis S. Greene, 1710 Rhode Island Ave. N. W., Washington.
Secretary: Dr. R. E. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. H. F. McDuffie, 45 Edgewood Ave., Atlanta, Ga.
Secretary: Dr. Leon E. Brawner, 157 Forrest Ave. N. E., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: 8 p. m., last Thursday of each month.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Samuel Salinger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Grill Room, Stevens Restaurant, 16 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

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Secretary: Dr. Robert J. Masters, 601 Hume-Mansur Bldg., Indianapolis.
Place: French Lick Springs Hotel. Time: Dec. 12-13, 1929.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Robert J. Masters, Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. M. Earle Brown, Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. W. A. Wagner, Pere Marquette Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., third Thursday of each month from September to June.

BALTIMORE CITY MEDICAL SOCIETY, SECTION ON OTOLOGY AND LARYNGOLOGY

Chairman: Dr. Waitman F. Zinn, Medical Arts Bldg., Baltimore.
Secretary: Dr. Moses L. Breitstein, 1212 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

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Secretary-Treasurer: Dr. Charles T. Porter, 520 Commonwealth Ave., Boston.

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Secretary-Treasurer: Dr. Don A. Cohoe, 13535 Woodward Ave., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

THE MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

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Secretary-Treasurer: Dr. J. H. Morse, 80 S. 7th St., Minneapolis.

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Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

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Secretary-Treasurer: Dr. J. C. Davis, 1615 Howard St., Omaha.

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Secretary-Treasurer: Dr. Donald Lyle, 19 W. 7th St., Cincinnati.
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President: Dr. J. E. Raia, 272 Broadway, Providence.
Secretary: Dr. F. W. Dimmitt, Jr., 195 Thayer St., Providence.
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Chairman: Dr. William Thornwall Davis, 927 Farragut Sq., Washington, D. C.
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Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

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President: Dr. John H. Foster, 1300 Walker Ave., Houston, Texas.
Secretary: Dr. Edward W. Griffey, Houston Clinic, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

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